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RETINAL GLIOMATA TREATED BY RADON SEEDS

BY

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THE following cases are reported in detail as a means of treating a condition for which it can be said there has been no alternative to removal of the eye, and when, as is frequently the case, both eyes are affected, the only remedy available in the past has been removal of both eyes, a mutilation to which parents will often refuse to submit their children. From the year 1871 to the end of 1924 there have been reported at the Moorfields Eye Hospital 163 cases of glioma of the retina, and of these the proportion of bilateral to unilateral cases was 1 to 5·11. (*Proc. Roy. Soc. Med.*, 1929, Vol. XXII, No. 9, p. 951).

It is proposed in the first place to describe in detail the clinical features of the five cases here reported, and afterwards to describe the technique, the choice of case, the changes which were observed to take place in the growths and tissues of the eye, and the results obtained.

Mr. Stallard alone is responsible for the preparation of macroscopic as well as the microscopic specimens, and for the examination and report upon all of these.

In two only of these five cases was it possible to watch, for any considerable period, the disappearance of the growths and the subsequent changes which took place in them.

In February, 1929, one of us had treated a case of sarcoma of the

choroid by insertion of radon seeds into the growth (*Brit. Jl. of Ophthal.*, Vol. XIV., p. 145), and now, two and a half years after, this patient is well, the growth remains shrunken, there is no extra-ocular extension, and no glaucoma; and there is no discoverable evidence that dissemination has occurred.

In the light of the above experience we took the first available opportunity of treating a glioma in the same way when it seemed justifiable, and this opportunity was afforded in October, 1929.

A small boy, E.H., aged 5 months, was seen by Mr. Rupert Scott, at St. Bartholomew's Hospital, on April 6, 1925, on account of the unusual appearance of the left eye. Glioma of retina was diagnosed and, three days later, the eye was removed. The diagnosis was confirmed by pathological examination.

On October 30, 1929, the child was again brought to see Mr. Scott because there was more discharge from the left socket, and it was said there had been pain in the right eye. Mr. Scott again took the opportunity to examine the right eye, and found a glioma at the upper and inner part.

Six children of the family were well; the first child was found to have had bilateral glioma of the retina at the age of a year, and died six weeks afterwards, apparently of intracranial extension.

Two children died of convulsions; the father died of a "tuberculous throat" in 1929.

On examination of the right eye a growth, evidently a glioma, was found in the upper nasal quadrant; the lower limits of it were well defined and here it was between three and four discs' breadth from the edge of the papilla. The upper edge was not easily seen and was not so sharply limited, vessels were seen in the tumour, and one or two small haemorrhages were present on the surface of it (Fig. 1).

Having regard to the experience of the first child, the mother refused to have the only remaining eye of this boy removed and, in these circumstances, we were glad to have the opportunity of treating the growth by radium, and Mr. Scott was good enough to transfer him to our care for this purpose.

The first seed was inserted on November 15, 1929; it was of 3 millicuries strength and, on ophthalmoscopic examination afterwards, it was found that no vitreous haemorrhage had occurred to obscure the view; every detail was seen as clearly as before, the steely blue seed was seen to have traversed the growth, and its point was projecting from its lower end, it having tunnelled the mass of growth a little too far on the temporal side; had it been a millimetre more in the lower nasal direction it would have been buried completely in the glioma and would not have shown on ophthalmoscopic examination; the apex of the seed was a little nearer the disc than the nearest point of the growth (Fig. 2).

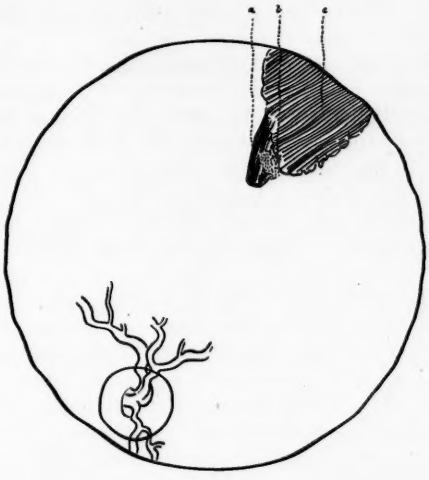

FIG. 2.

Observations of the growth and the changes that took place in it were easily made, and there has been no difficulty in this regard up to the present time; the changes will be referred to in detail under the appropriate head.

On November 26, *i.e.*, ten days after the insertion, the seed was removed: again the media remained clear and, immediately after the removal, the hole through which the seed had been introduced was clearly seen, as a dark grey crater, having a sharp cut falciform upper edge, and shelving off below to the level of the rest of the fundus, with a small fringe of haemorrhage attached to it (Fig. 3).

There was no retinal detachment or other gross change except in the immediate locality of the growth.

Detailed observations of the growth were made.

By December 3, it was clear that the growth was less bulky and was shrinking; this was particularly noticeable in that the point previously nearest to the disc had perceptibly receded.

The boy was discharged from hospital on December 8, 1929, He was shown at a clinical meeting of the Royal Society of Medicine on December 13, 1929 (*Proc. Roy. Soc. Med.*, Vol. XXIII, No. 4, February, 1930, p. 475). He was re-admitted for observation from January 17, 1930, to February 19. We were particularly anxious to assure ourselves as to whether there was any recrudescence of the growth. We, however, came to the conclusion that none was present, and he was discharged.

On July 6 he was re-admitted because we thought that there was a recurrence of the growth in the region of the hole of insertion;

there was no certain sign of this and, looking back, we think perhaps we were too apprehensive.

On July 8 a fresh seed of 5·3 millicuries strength was inserted through the same hole as the previous seed—there was no constitutional disturbance. On the next day it was found that the seed had slipped entirely within the globe, so that a further operation was necessary to fix it within the hole. Certain changes occurred in the retina at a distance from the hole, which are referred to later, as the result of this accident, but there was no haemorrhagic or other disturbances of the vitreous, and every detail of the fundus was as clearly seen as before.

This second seed was removed on July 15, *i.e.*, a week after its insertion. No difficulty was experienced in finding it and removing it and the intra-ocular appearances were little changed as a result, except that a hole replaced the seed (Fig. 3). The patient was seen on January 2, 1931, when the intra-ocular condition seemed to be stable, with no evidence of an active growth in it; he was attending school with other children, keeping well, and putting on weight.

He was next seen on March 13, 1931, when he was shown at a meeting of the section of the Royal Society of Medicine, and it then seemed certain that changes had occurred which were not of the nature of scarring, but were indicative of recrudescence at the edge. He was therefore re-admitted for further treatment, the method of which is described later.

He was examined on May 15, 1931; after this further treatment was completed. No alteration in the intra-ocular appearances was appreciable, his visual acuity was at least 6/12, the lens and media were unaltered. The retina had a faintly mottled appearance in places, but we could not be sure that it represented a pathological change. The mother spontaneously stated that so soon as it became at all dusk he saw less well than her other children. He was last examined by all of us on September 11, when it was evident that changes had occurred.

As regards the site of the growth; it was completely scarred and there was no sign of the neoplasm, so that it may be said with certainty that the original glioma had completely disappeared and remained scarred after so long as a year and ten months. One of us, however, was not quite sure that there was not a recurrence at the temporal edge.

Other changes had, however, developed in the optic disc. Its edges were blurred, a number of flame-shaped haemorrhages had developed around it, several pale areas were present, and the sight had deteriorated. The child was ill looking and frequently awoke at night in an attack of "terror."

It seems likely that the disc changes indicate an intra-cranial deposit although the appearances are not typical of papilloedema,

and alternatively they probably imply local retinal deposits: the future progress will probably make it clear which of these is the case. There still was a tendency to redness of the skin of the lids and lid margins.

Case 2.—Winifred B., aged 5 weeks. Two weeks after birth the mother noticed "a redness in the middle of the right eye." Two weeks later the child attended Moorfields, under the care of Mr. P. G. Doyne, and was admitted for a staff opinion, and no doubt was expressed by any member of the staff that the condition was a glioma. Mr. Doyne, knowing of our interest in such cases, was good enough to transfer her to our care.

This child was the seventh child in the family and all the rest were normal. She was born at full term, and there was no history of glioma in the other children or the parents.

There was a white mass filling the vitreous chamber. It had a nodular surface with a number of dilated vessels on it, no normal fundus was visible, the cornea, iris, anterior chamber, and lens were normal. There was no clinical evidence of extra-ocular extension. The left fundus was examined under a general anaesthetic, and no growth was found in it.

On October 31, 1930, one radon seed of a strength of 3·5 millicuries was inserted into the growth, through the lower temporal quadrant and behind the equator: the wound healed without serious reaction and on the tenth day, after a further exploration of the left eye, and having made as certain as possible that no growth was there, it seemed best to remove the right eye.

This was done on November 11, together with the seed *in situ*, and four seeds of 3·5 millicuries strength were put into the orbital tissues at the same time, as a precautionary measure.

During the ten days that the seed was in the eye there was no appreciable change in the appearance of the growth, the lens and cornea remained clear, the anterior chamber was well formed, and the iris was normal.

For twelve hours after the operation the temperature was 100°F., P. 155, and R. 46, but they all subsided to normal: the lids were slightly swollen and reddened.

The seeds in the orbit were removed a week later: the socket continued to discharge a turbid, sanious fluid, and the surrounding skin was reddened. The detailed report is included later.

The child was examined again on March 3, 1931, under general anaesthesia, to be assured that no growth was present in the left eye, and none was found after a careful search. She was healthy and well and now making good progress.

Case 3.—Albert P., aged 3 months, was seen at the Royal Eye Hospital, Manchester, on September 8, 1930, by Mr. Milnes Bride, who diagnosed bilateral retinal glioma. He removed the left eye,

which was the worse of the two, and confirmed the diagnosis by pathological examination, and was then good enough to refer the child to us.

The patient was a healthy looking boy, the left socket was clean. The right cornea was clear, the pupil was active, and the iris normal: there was a large mass of glioma occupying the greater part of the temporal half of the fundus (*a* Fig. 4) and, what appeared to be a simple retinal detachment below (*b* Fig. 4).

On September 23, two seeds of a 4·8 millicuries strength were inserted into the glioma. There was a large mass of growth,

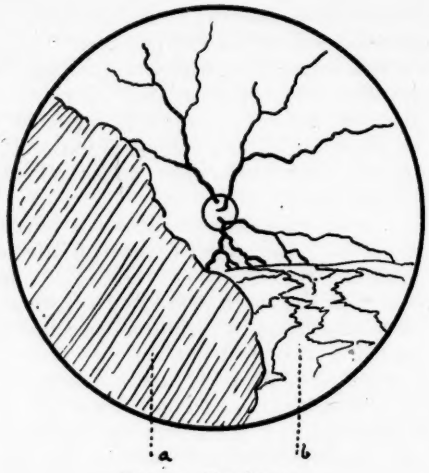

FIG. 4. October 23.

evidently sufficient to embrace completely the seeds. They were therefore inserted free into the mass of the growth, without any fixing collar.

After inserting the seeds it was found, on ophthalmoscopic examination, that one of them was too far forward, so that a considerable part of it was protruding beyond the growth into the vitreous; it was therefore removed and re-inserted further back, so as to be embedded almost completely within the growth.

There was a slight rise of pulse rate after the operation.

Ten days later, on October 3, the seeds were removed without trouble, and it was then noticed that changes had developed in the lens; these changes are referred to in detail under a separate head.

Two weeks later, on October 16, the eye was examined under a general anaesthetic. There was redness of the skin of the lids and

FIG. 1.

FIG. 3.

FIG. 6.

surrounding tissues, but of no marked degree. All fundus details were clearly seen, there appeared to be some loose fragments of growth below; the main mass of the growth was readily seen, and was believed to be smaller. The child continued to improve physically and to increase in weight, having gained 2 lbs. 2 oz. since admission, *i.e.*, five weeks.

Examination under general anaesthesia was again effected on November 18, when it was clear that the mass of growth was

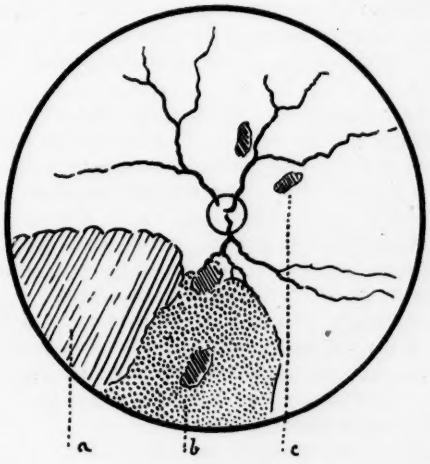

FIG. 5. December 2.

shrinking (*a* Fig. 5); the detached portion of the retina had a degenerate appearance with pale pigmented areas in it (*b* Fig. 5), and three islets in outlying districts were present, which might well be gliomatous deposits (*c* Fig. 5).

On December 2, examination again revealed little alteration.

On January 2, 1931, the child was again examined under a general anaesthetic. It was not possible to appreciate any alteration in the islands of glioma, but it seemed that the main mass was increasing at one part.

A 3·5 millicuries seed was inserted through the old hole, using a rubber fixing shield to hold it in position. Next day the child appeared to be suffering from a chill, but the local progress was satisfactory till the 8th day after operation when, as explained later, panophthalmitis developed.

Case 4.—This child had both eyes so full of growth that no retina that was certainly normal could be seen in either eye. It has almost no value, except with regard to the technique of insertion, to

which reference has been made. The child died on the table, of massive collapse of the lungs, just after the insertion of seeds in both eyes was completed.

Case 5.—E. C., aged 1½ years.

The parents had noticed at the age of two months that the left pupil was "light in colour," but did not seek advice till seven months later when they took the child to the Royal Eye Hospital where glioma was diagnosed and the eye removed.

Six months later the parents believed the sight of the right eye was not good, and glioma was again diagnosed, but it was suggested that, before sacrificing the second eye, they should obtain a confirmatory opinion and, with this in view, they attended Moorfields on June 25, 1931.

On examination under an anaesthetic it was evident that the eye was largely filled by growth, and a small area only of what appeared to be normal retina was visible. An endeavour was made to make the case clear to the parents, and at their request radium was used.

On June 27 four seeds of 3·5 millicuries were applied, as nearly as could be judged at equal distance from each other, two intra-ocularly and two in contact with the sclerotic.

On ophthalmoscopic examination immediately afterwards it was seen that the two intra-ocular seeds were successfully buried in the growth and the only disturbance produced by their insertion, as judged by the ophthalmoscope, was the presence of two or three small areas of haemorrhage.

All the seeds were removed ten days later. A good deal of reaction developed in the conjunctiva and in the substance of the lids, making examination of the eye unsatisfactory, and no certain change in the growth was observed with the ophthalmoscope.

The eye was removed on August 4. The child recovered and put on flesh, and when last seen on September 28 was in good condition; the swelling of the lids had almost completely subsided; the lids were, however, still very red.

For Mr. Stallard's report, see later.

It will be agreed that for so desperate a condition as bilateral glioma of the retina a form of treatment which may be considered experimental needs no justification as an alternative to enucleation of both eyes, if it offers any reasonable hope of success: indeed many parents will refuse to submit their children to so dreadful a mutilation, although no doubt such an individual, in after life, can have and can form no sort of idea of what it is that is missing to him.

In the *Proc. Roy. Soc. Med.*, Vol. XXII, No. 7, p. 951,

1929, one of us published two of the earliest possible cases of retinal glioma: in each the diameter of the growth was a little greater than that of the optic disc (Fig. 6).

In the one case, on the advice of colleagues at Moorfields, we told the parents that the best prospect, from the point of view of the child's life, was to have both eyes removed; and to this they submitted. In the second case treatment of any kind was refused.

In the light of our present experience we should not again entertain the immediate removal of such eyes, nor consider it a proper procedure.

Choice of Case.—In cases of bilateral glioma there will be no divergence of view, we think, with regard to the desirability of some form of radium treatment, for one eye at least, whenever there is any reasonable area of intact retina. In cases, however, like case 4, in which it is clear that even if it could be certain that the glioma would be cured, yet there is no chance of vision being obtained, enucleation is clearly called for.

The case, however, is quite different where, after the most careful search under general anaesthesia, the second eye is proved to be free of growth; and most surgeons would advise removal of the affected eye in such circumstances.

From the nature of things one seldom sees an early glioma of the retina, except where it is discovered in the second eye as a result of the child having been brought for a glioma, which has given rise to signs which have been observed by the parents in the first eye, signs which, therefore, imply at least a somewhat advanced growth.

It seems likely that, so long as it can be seen that the optic disc is not involved by the growth, there is little or no danger of dissemination, and experience may perhaps prove that the insertion of radon seeds in unilateral cases is a proper procedure if a considerable area of normal retina be present. This, however, remains for future decision.

Technique.—The seeds we have used have all been prepared for us by Prof. Hopwood, who is in charge of the radium department at St. Bartholomew's Hospital.

Strength of Seeds.—Forty seeds have been inserted in various ways—the weakest strength used has been 1 millicurie, and the strongest 5·3 millicuries, and in 11 instances the strength has been approximately 3·5 millicuries; all have been filtered through 0·5 mm. of platinum. Figure 7 shows a seed with the silk attached alongside a millimetre rule.

Technique of Insertion.—Our endeavour has been so to insert the seed that it is completely embedded in the growth, so that if possible no seed is seen on ophthalmoscopic examination afterwards; twice only have we succeeded in doing this, on the other occasions a part of the seed has been visible.

On two occasions we have used a local anaesthetic, a general anaesthetic has been the rule, but local injections are preferable when at all possible.

The first point of importance is to locate the growth, so that the seed is completely embedded in it; and for marking the radius along which it lies one can make use of the method employed for locating the hole in cases of detached retina. The distance from the limbus and from the disc can best be judged by means of a general survey and consideration of the position with respect to the disc and the

FIG. 7.

ora serrata. It will be found that the general tendency is to judge the growth to be further forward than it is.

Exposure of the Sclera.—There are several ways of reflecting the conjunctiva and Tenon's capsule for the exposure of the sclerotic, and there are two chief desiderata; first to give easy access, and secondly, we believe it is important that the reflection is so planned that the hole in the sclerotic is well distant from the incisions through the conjunctiva, so as to make the spread of infection into the globe along the track of the silk as unlikely as possible.

Where the incision into the eye is to be made at all in front of the equator (*b* Fig. 5), we believe the flap indicated in Fig. 8 is best, and it is a good plan to have the stitch at *a* put in beforehand, so that it is all ready for tying afterwards. When the point of the insertion is to be further back, a similar shaped incision, but placed further back, gives good exposure and good covering afterwards—in any case a large flap is best. A strabismus hook or a small double sharp hook form convenient retractors for the soft tissues, and the globe can be pulled into the desired position by means of a stitch

passed through the episcleral tissues at the requisite spot, or by means of a fine sharp double hook which catches on to the sclera; or a liberal and firm forceps grip of the deep surface of the soft tissues, where they have been reflected from the eye, may be taken as far back as possible; it will be found that these tissues can be dragged upon forcibly and the eye rotated into the desired position, by this means, without there being any pressure put upon the globe.

The Incision.—The incision through the sclera is made with a broad cutting needle; and sometimes one can get an indication as

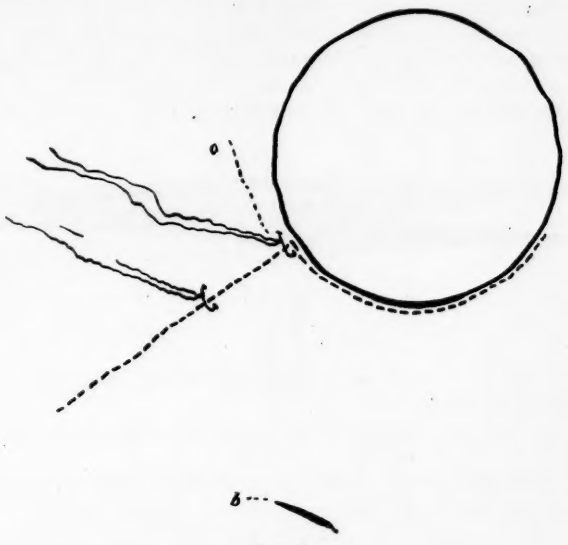

FIG. 8.

to the best direction for the track, for clearly, in the case of a sarcoma at any rate, the ideal track will be a perpendicular directed inwards from the centre of the base of the growth and so an endeavour is made to insert the cutting needle in this direction and, with this in view, we have had needles made bent at right angles, and at an obtuse angle.

It has seemed best that the hole made in the sclerotic should be of such a size as just to transmit the seed and no larger, and Messrs. Weiss have therefore made the blade of the needle 3 mm. across, which width is suitable for the standard sized seed (Fig. 7); the needle is dipped into a solution of gentian violet and allowed to dry; by this means the cut edges of the incision are stained and so the point for the introduction of the seed is marked.

It seemed advisable that a track should be deliberately cut in the substance of the growth, into which the seed is to be introduced, otherwise on inserting the blunt pointed seed the growth may be pushed in front of it instead of being transfixed by it; this has happened in one case at least, and we believe that the end is best attained by inserting the needle well into the growth thereby cutting a track in it into which the seed is introduced.

The seed is now taken in the introducing forceps and inserted, with a slight wriggling motion, through the hole in the sclerotic. This forceps is made with blades at any angle corresponding with the angle of the needle which has been used, so that the seed is inserted in the line of the track made by the needle (Fig. 9).

If the growth is of considerable size the seed is pushed right home by means of the dimple made for the purpose on the side of the heel of the forceps, and the silk attached to the end of it is now

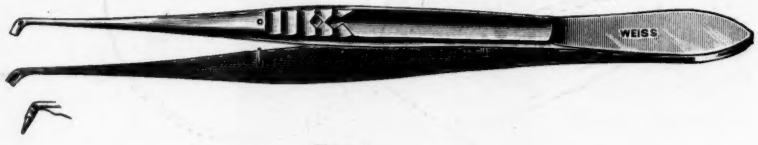

FIG. 9.

cut short leaving a length of about 1¼ inches, by means of which the seed is removed later.

The silk should be black in colour so as to facilitate the finding of it when the seed is to be removed.

The conjunctival flap is now replaced and sewn in position, the short piece of black silk being completely buried under it.

Some differing degree of difficulty may arise when it is necessary to insert the seed far back, a difficulty that varies with the prominence of the eye, with the width and looseness of the palpebral fissure, and especially with the point of insertion.

It may be said that there is no great difficulty in getting as far back as the equator anywhere in its circumference, except perhaps above, and that on the outer side one can get further back without difficulty.

In Case 1, the apex of the seed was within 3 disc breadths of the edge of the disc and there would have been no difficulty in getting it further back so as to be in contact with the disc.

In Case 4, the point of insertion was about 5·5 mm. from the disc, and Mr. Stallard has measured the nearest point to which the sclera had been cleared and it was 1·5 mm. from the disc.

We have avoided dividing a muscle as a preliminary because of the necessary disturbance of the replaced muscle at the subsequent

removal of the seed; we should not, however, hesitate to do it, and in some situations it would clearly be necessary and would be a great help; and, further, we fancy, it would not be difficult to replace the muscle after the insertion, bringing the silk attached to the seed through its substance so that the seed might be removed afterwards without a second resection.

In some cases the mass of growth may be insufficient to embrace the seed and hold it in position, so that it may fall loose inside the globe. This happened in Case 1 where the second seed was inserted as a precautionary measure when the bulk of the growth had disappeared, so there was insufficient mass to embrace it. In such a case we have used a very small rubber shield, which can be boiled, through the centre of which a small hole is made, and into which

FIG. 10.

the seed is inserted so that its hind end is held in the ring and, as an additional precaution, the silk of the seed is threaded through the substance of the rubber (Fig. 10).

The position of the part of the seed within the globe can be planned according to the direction of the track of the hole through the rubber washer.

Where it has seemed advisable to put in a second seed at a later date there has been no difficulty in finding the old hole of entry, by its dark colour and, in Case 1 even after seventeen months, it was quite easily identified.

Removal of the Seed.—It was planned to keep the seeds in for ten days in every case, and this rule has been carried out with very little variation.

The original conjunctival incision has been opened up, the conjunctival flap turned back again, the black silk found, the hole exposed, and the seed extracted by pulling on the thread.

In no case has there been any complication arising from this, such as haemorrhage into the eye or externally, nor any loss of vitreous,

and although one has felt a little apprehensive lest the silk should snap at its attachment to the metal, it has not happened.

Changes which occur as a result of the Seed.—On the first occasion that a seed was inserted in February, 1929, we wondered whether necrosis of the sclerotic might occur immediately around, but it, and all succeeding cases, have shown that there is no fear of this with the strongest seeds. After the introduction there has been reaction of the outer parts of the eye, but for the most part not of excessive degree, no undue chemosis and, apart from the case in which infection occurred, there has been very little evidence of intra-ocular reaction; in no case has there been evidence of cyclitis; in each of two cases a single posterior synechia has formed. A rather common occurrence, however, has been what perhaps may best be described as an eczematous condition of the lid margins and adjoining skin overlying the site of insertion. and this has been long persistent; it was severe in Case 5.

In four adult cases in which seeds have been inserted special inquiry has been made as to the occurrence of pain: one complained of some, one said he suffered short attacks of knocking sort of pain, at about twelve hour intervals, but of no particular severity; the third man said he at first had a bout of twenty minutes of pain a day, on the sixth day it became quite severe for some hours, and persisted after this period till the seed was removed. Following this there was no pain. The fourth man had a good deal of pain.

With these four cases in mind, and judging from the behaviour of the children who were operated upon, we think it probable that these children suffer no very severe pain from the procedure.

On examining with the ophthalmoscope, while the patient was still on the table, in no case so far has the view of the interior of the eye been spoiled or even obscured by haemorrhage; thus, in one case, it was not possible to see the slightest change of any kind, it was exactly as if nothing had been done. In other cases the light, steely blue apex of the seed has been seen protruding from the area of growth, or showing through the intact retina.

In Case 1, November 15, 1929, on examining with the ophthalmoscope immediately the seed was inserted a filmy sort of cloud was present, partly overlying the exposed part of the seed; this we took to be coagulated vitreous (*b* Fig. 2).

Nineteen hours later this had increased so as further to hide the seed, irregular streaks of blood were present on the surface, and a band of blood was present, apparently trickling along under the edge of the seed. Three days after the insertion all details were seen as clearly as before the operation, there was no evidence of infection, the exudate overlaying the seed became more solid looking, and the seed was still fringed by haemorrhage. On November 22 it seemed that the exudate was clearing, and the whole growth had

an opaque, white, rather woolly appearance, and four days later the exudate had almost gone.

The seed was removed on November 26, no vitreous haemorrhage or retinal detachment resulted, and the hole of entry was plainly seen as a clear-cut, greyish, deep recess (Fig 3).

By December 3, *i.e.*, eighteen days after insertion, it was possible to say that the growth was disappearing; the part protruding towards the disc had, in considerable measure, disappeared, an area of dark haemorrhage was still present, and there was a small patch of pigment to the outer edge of the hole, which has remained since.

The boy was discharged from hospital on December 8, 1929, and was re-admitted for observation on January 17, 1930, when the condition was as seen in Fig. 3.

On April 15, the note was, "I do not think that there is any fresh change except that it seems to me probable that the hole is filling up, and the edges are perhaps thickening as if from new growth, but I do not think the change is sufficient to be sure of it."

July 8. A fresh seed of a strength of 5·3 millicuries was inserted through the old hole, as there was a suspicion that a recurrence was taking place at the upper margin of the hole. This seed slipped free into the vitreous, but was recovered and fixed as described under the head of Technique.

July 23. There were some strands of vitreous opacity attached to the lower part of the hole. A little fresh haemorrhage occurred at the upper edge, and a little white exudate and strands of vitreous were observed attached to the seed. There was no constitutional disturbance, and the clarity of the vitreous was not impaired.

Four days after the insertion of this seed three small patches of exudate were present along the upper temporal vessels and one larger patch along the upper nasal; events seemed to substantiate the fact that these were a result of the seed dropping into the vitreous.

Next day two fresh areas were seen and Mr. Stallard observed that all these areas had shifted downwards, thus showing that they were in the vitreous.

This seed was removed on July 15. Eight days later the opacities in the vitreous had completely disappeared, a few haemorrhages were present at the edge of the hole and elsewhere, and strands were present in the vitreous.

Below the sharp falciform upper edge of the hole was a deep blue grey recess, and this shelved off below into the retina, which had a mottled appearance.

There was a small fluffy mass which was marked to be watched for recurrence, and as accurate and detailed drawing as possible was

made of the upper edge of the hole, also with a view of watching any change in it.

On November 2.—The note was: "a few bright dots in the surrounding retina, now no haemorrhages and indeed I should be unable to be sure of any change at all as compared with the appearances before the seed was put in in July."

December 30, 1930.—He was seen by Mr. Stallard, who noted that the hole was filling in, apparently by fibrous tissue. A strand of vessels was extending into the vitreous from the nasal edge of the hole, but the pigmented area of the retina was unaltered.

On March 13, 1931.—The boy was again shown at the Ophthalmological Section of the Royal Society of Medicine. He was in good health, was attending school, and had an acuity of 6/9 for certain. The edges of the hole, above and to the temporal side, were more solid looking, and the vascular strand extending forward into the vitreous had increased, and we believed at these points there was a recurrence, although the changes were slight.

On March 20, therefore, under general anaesthesia, a conjunctival flap was reflected, as at the first operation, and the point of insertion through the sclerotic of the previous seeds was found. It was easily identified as a dark, broadly linear marking, quite flat and smooth, with no suggestion of any growth or other tissue protruding through it.

Four seeds of 3·5 millicuries strength were applied to the sclerotic in such a way that the hole was, approximately, in the centre of them.

We had a fine groove filed in the platinum coating around the middle, and around this groove fine silk was tied, by means of which the seed was sewn in position on the sclerotic (Figs. 11 and 12).

The conjunctival flap was replaced. The boy complained of no pain afterwards, nor was there any unusual degree of reaction; the lid margins and adjoining skin showed the rather common eczematous condition.

After ten days the four seeds were removed without difficulty, and the conjunctiva replaced in position.

Observations on the growth are continuing (see earlier). It may be that this, or some similar form of contact application, will prove as efficient as intra-ocular insertion. We have at present under observation two other cases in which seeds have been applied in this way, but for the time being we have no sufficient knowledge on this point.

In Case 2.—All that one can say about the appearance of the growth is that the insertion of the seed was carried out without at all obscuring the view of the intra-ocular tissues, and that, during the ten days that it was under observation, no appreciable change

was noticed in the growth, and that no lens changes or evidence of intra-ocular inflammation occurred, nor was there coagulation or other change in the vitreous.

It was hoped that some definite histological change might be demonstrated in the cells of the growth as a result of their ten days' exposure to the emanations, and Mr. Stallard's report, which follows, is of interest in this connection.

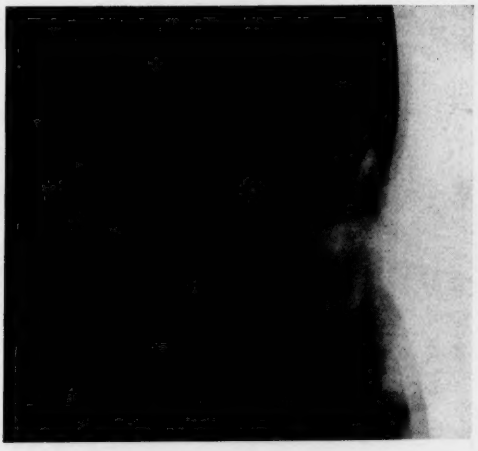

FIG. 11.

FIG. 12.

Case 2.—Pathological Report. Macroscopic Appearances. The glioma is mainly exophytum, the retina being completely detached and lying in a folded state just behind the lens. The radon seed (see Fig. 13) is embedded in the lower half of the glioma, and its point is apposed to the posterior aspect of the detached retina at six o'clock and in the same plane as the equator of the lens.

The neoplasm for 3 mm. around the seed shows macroscopic evidence of degenerative changes. This area is homogeneous and structureless and has several small scattered haemorrhages. The upper part of the glioma at a greater distance than 3 mm. from the

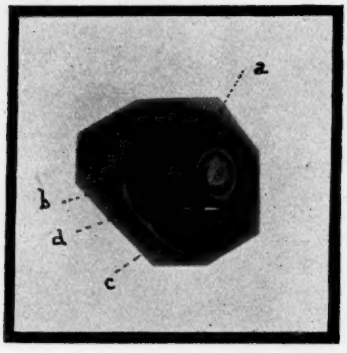

FIG. 13. CASE 2. Winifred B., aged 5 weeks.

Photograph of globe divided vertically and to the temporal side of the optic nerve. The globe is embedded in celloidin.
a Detached retina. *b* Glioma retinae. *c* Radon seed. *d* Degenerate area of growth in the vicinity of the radon seed.

seed retains the granular "cream-cheese" appearance which is characteristic of this neoplasm.

The choroid is oedematous and there is some exudate in the supra-choroidal lymph space and the anterior chamber.

There is no macroscopic evidence of extra-ocular extension of the glioma.

Microscopic Appearances. (Preparation—Zenker fixed. Embedded in celloidin. Serial sections down to the site of the radon seed. Stained with haematoxylin and eosin).

The retina is completely detached from the optic disc to the ora serrata, the line of cleavage extending a short way between the pigmented and non-pigmented epithelial layers of the pars plana. Degenerative changes, oedema and small haemorrhages are present in the retina. The retinal vessels are congested.

The glioma arises from the inner nuclear layer and extends into the sub-retinal space. Degenerative changes are more marked in the vicinity of the radon seed, but are also present throughout the whole neoplasm. The tumour cells are variable in size and shape; some are swollen and others shrunken and crenated. The chromatin network is broken up into minute spherules which are disposed in an irregular manner in the cells, and in some cases project from the periphery of the cell as minute excrescences. The cells in which degeneration is more advanced stain pink with eosin and the nuclear remains are represented by some faint blue dots (Fig. 14).

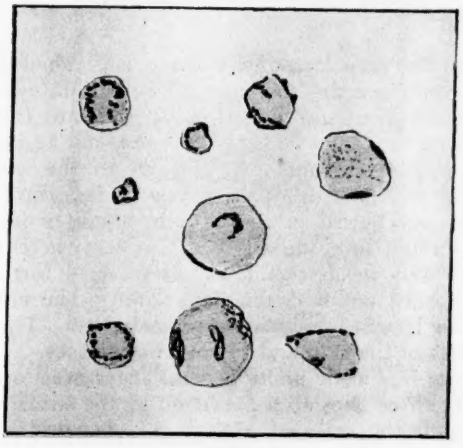

FIG. 14.

Diagram of some of the glioma cells which have undergone changes as a result of exposure to radium emanations.
Necrobiosis, chromatolysis, granulation and vacuolation are represented.

Degenerative changes are present throughout the neoplasm, but in the upper part there are a few islands of glioma cells the viability of which is doubtful. As these islands do not possess a central capillary and are presumably cut off from any blood supply, it may be assumed that their destruction is in progress. The capillaries which permeate the growth are dilated and surrounded by a narrow zone of degenerate glioma cells. Glioma cells extend up to the edge of the optic disc, but have not infiltrated the optic nerve.

Irregular clumps of brown pigment are scattered sparsely through the neoplasm, and haemorrhages are also present.

All the vessels of the uveal tract and the capillaries in the optic nerve are congested. There is oedema of the choroid and exudate in the supra-choroidal lymph space and the anterior chamber. The

anterior two-thirds of the lens showed no change in structure (the posterior portion fell away in the process of section cutting.)

Conclusions.—The nature of the neoplasm was proved to be a glioma retinae exophytum. It is probable that the entire tumour was destroyed by the radium emanations and that growth was arrested. The destructive action is particularly well marked in a zone 3-4 mm. around the radon seed in all directions. Except for some localized oedema and congestion of the vessels of the uveal tract the radium emanations do not appear to have had an adverse effect on any of the other structures of the eye, so far as histological appearances are concerned.

Case 3.—In this case Mr. Milnes Bride had removed the worse eye and had confirmed the diagnosis. In the right eye was a large mass to the lower temporal side (Fig. 4), and into this two seeds were inserted on September 23, 1930; one about 15 degrees to the south of west, and one about 15 degrees to the west of south. On examining with the ophthalmoscope it was seen that the top seed was not well buried in the growth, a half of its length was protruding through into the vitreous. It was therefore removed straight away and without difficulty re-inserted further back.

The seeds were removed ten days later. The changes which occurred in the lens are discussed separately later. There was some general redness of the skin and surrounding tissues.

Examination was made under general anaesthesia on October 16, that is twenty three days after insertion of the seeds. Everything was clearly seen, the mass of growth was believed to be smaller, though one could not be sure of this, and some loose fragments of growth were scattered about on the retina.

On November 7, *i.e.*, forty-five days after insertion, the growth had altered in appearance, having a "cream cheese" sort of appearance; a single synechia had developed. The child still increased in weight.

On November 18, the glioma was shrinking, and the adjoining retina was taking on the degenerate appearance which occurred in Case 1. On December 2, the growth was further reduced in bulk, and the area of degenerate retina was increased (Fig. 5). The small outlying areas in the retina were little altered.

On December 12, the child was examined again under anaesthesia, and a small fragment which overlaid the point of insertion of the seed was removed for examination. It proved to be fibrous and granulation tissue only.

The isolated fragments did not increase in size, but it appeared that one part of the growth was increasing; a 3·5 millicurie seed was therefore inserted on January 21, 1931, held in position by a

rubber washer; and a new departure was to leave the silk attached to the seed long, bringing it out through the palpebral fissure and securing it to the cheek.

The next day the child showed signs of bronchial catarrh with nasal discharge, and soon became rather ill of it, and was too ill for anaesthesia to be advisable.

On the thirteenth day the seed was removed. Streptococcus haemolyticus was found in cultures of the conjunctival sac, and it soon became apparent that panophthalmitis was present, and

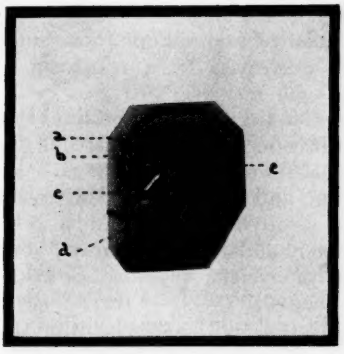

FIG. 15. Case 4. R.M., aged 6½ months.

Photograph of globe divided obliquely. The specimen is embedded in celloidin.

a Glioma. *b* Choroidal haemorrhage. *c* Radon seed inserted 5·5 mm. from the temporal edge of the optic disc. *d* is the most posterior point to which the sclera was exposed at operation. It is 1·5 mm. from the temporal edge of the optic disc. *e* Sub-retinal exudate.

ultimately the eye was removed. The child recovered and left hospital seemingly happy and well and in good health.

Case 4.—Fig. 15 shows one of the eyes with a seed *in situ* and, as will be seen, it was found possible to insert it at a point within 5·5 mm. of the optic disc. The sclera was found to have been cleared to a point 1·5 mm. from the disc, at which point a seed could easily have been put in, had it seemed advisable.

Case 5. *Macroscopic Appearances.*—The globe is small and shrunken.

There is a corneal ulcer which has perforated, and the remainder of the cornea is thin and ectatic. The sclera is thickened. There is no macroscopic evidence of extra-ocular extension of a neoplasm. The eye was divided horizontally, and the advance of the knife was arrested by a dense calcareous mass which occupied the vitreous

chamber. The anterior chamber is absent, the vitreous lying forward, and the retina detached.

Occupying the vitreous chamber is a neoplasm in which are calcareous deposits. The choroid is oedematous and congested.

Microscopic Appearances.—The corneal ulcer has perforated and the lens capsule is apposed to the posterior aspect of the cornea. The iris is converted into granulation tissue, and presents many new vessels which are congested. The vitreous has been forced forward and contains leucocytes, red blood corpuscles, and exudate.

The choroid is oedematous; the vessels are congested; there are some small haemorrhages and mild leucocytic infiltration is present. It is not invaded by the neoplasm.

The retina is detached and, except for a small strip at the ora serrata, is entirely destroyed by a neoplasm having the typical appearance of a glioma retinae.

The growth has extended into the subretinal space. Its diameters are 7·5 × 7·5 mm. It is degenerate; calcareous deposits are present; there is much fibrosis, and hyaline change. Haemorrhages, mild leucocytic infiltration, and congested blood vessels are in evidence. Except for twelve small islands of glioma cells, each measuring 1/10 mm. in diameter, and arranged around the periphery of the main mass of the neoplasm, histological evidence points to its devitalisation and necrosis.

The retina is infiltrated by the neoplasm up to the optic disc, but there is no evidence of invasion of the optic nerve. The optic nerve is atrophic and shows an increase of neuroglial tissue and dilatation of the capillaries. There is no histological evidence of extra-ocular extension.

Pathological Diagnosis.—Glioma retinae.

Conclusion.—Except for the twelve small islands of glioma cells seen over a series of sections the neoplasm has been destroyed.

Lenticular Changes.—In a previous paper (*Brit. Jl. of Ophthal.*, Vol. XIV, p. 145. 1930), one of us showed that an unusual star-shaped opacity in the posterior cortex of the lens disappeared under the influence of radon seeds. Changes of especial interest took place in the lens of Case 3.

On September 23, 1930, two seeds of a strength of 4·8 millicuries were inserted into the eye. These were removed ten days later and it was then observed that three groups of what appeared as aggregated vacuoles were present in the posterior cortex of the lens.

Thirteen days later these changes had increased so that now there were seven of these groups and, exactly as before, they seemed to be aggregations of transparent vacuoles in the lens substance (Fig. 16).

On November 7, *i.e.*, twenty-two days after the last examination, and forty-five days after the insertion of the seeds, five groups only of vacuoles were left (Fig. 17).

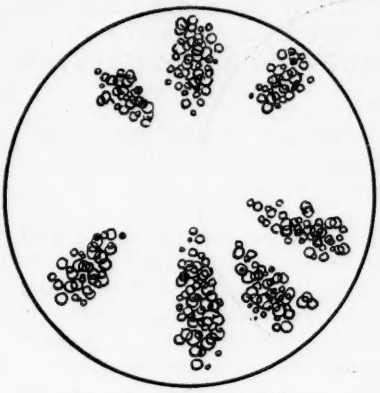

FIG. 16. October 14, 1930.

FIG. 17. November 7.

On November 18, there were still five groups (Fig. 18).

On December 2, four groups could be identified, though they were very small (Fig. 19) ; and on January 2, 1931, the whole lens had returned to its former clear condition, except that one small group was discernible in the upper part of the lens (Fig. 20).

This was the last occasion of observations on the lens.

It is not obvious why lenticular changes should have developed in this and not in the other cases, but one may recall that two seeds of 4·8 millicuries were inserted and, as remarked elsewhere, one seed was at first too far forward, that is, rather close to the lens, but it was not retained in the position for more than ten minutes, at most, before its removal.

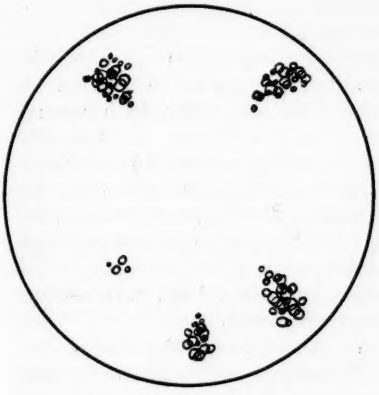

FIG. 18. November 18.

FIG. 19. December 2.

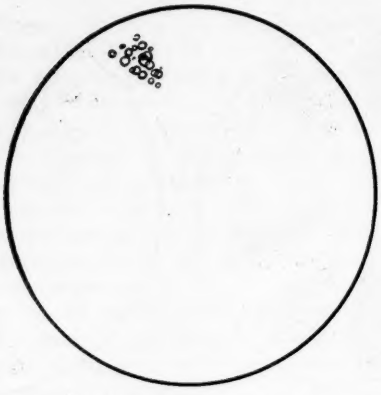

FIG. 20. January 2, 1931.

The increase in the opacities, which continued for some time after the seeds had been removed, is interesting, and especially also the fact that they disappeared practically completely within rather less than three months.

Conclusions.—It is evident that the data that are here presented are too scanty for much that can be properly set under the head of conclusions; sufficient, however, has been shown to guide us in further investigations, and enough it may be thought to justify further treatment along these lines, more especially in bilateral growths.

It is demonstrated that a seed of at least 5·3 millicuries may be inserted into the eye, and that at least five seeds, each of a strength of 3·5 millicuries, may be applied to the outside of the sclera without causing sloughing of any of the tissues.

The technique of insertion and subsequent removal of the seeds has been described and has been found to present no difficulties.

It is shown that a retinal glioma will disappear under the influence of radon seeds, and that in a concrete case a glioma of moderate size not only was not increased in size twenty-two months after the commencement of treatment, but was shrunken and seemed to be completely scarred; it is, however, premature to say that it is cured.

Histological changes in the glioma cells which have been exposed to the radon seeds have been demonstrated.

Opacities of a special type in the lens have been watched to develop and afterwards to disappear completely.

Further investigations with radon seeds on sarcomata as well as gliomata are being prosecuted, both intra-ocularly and as surface applications to the sclerotic.

THREE CASES OF RETINITIS PSEUDO-NEPHRITICA STELLATA. CONSIDERATIONS ON THE PATHOGENESIS OF NEURORETINITIS IN GENERAL

BY

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IN a previous paper we have discussed the different explanations proposed for the pathogenesis of so-called retinitis albuminurica. Based on 19 personal observations, we have adopted the opinion that the disease should be called the neuro-retinitis of Bright's disease, in order to point out first the constancy with which the papillary lesions are found and secondly that there is not such causal relationship between retinitis and nephritis as the usual name would seem to imply. In fact Bright's disease seems to be not a simple kidney process, but a more general metabolic disturbance touching at the same time, independently, kidney, retina and other territories in the body.

The discussion about this problem has continued actively during the last few years and although the most authoritative opinions are pretty close, a real understanding of the pathogenic mechanism is yet wanting.

The older theories which tried to identify different toxic products, accumulating in the body as a result of kidney insufficiency, and causing the retinitis, could not be verified. The most popular were those of Widal (azothaemia) and of Chauffard (cholesterinaemia). In the same line Poyales has recently pointed once more to the hypercreatininaemia.

The most authoritative workers, however, tend to-day to admit the direct, immediate influence of local circulatory troubles in the production of the retinal lesions. That such troubles are themselves due to some toxic influence, of renal or general origin, remains very probably true, and we shall return to that point later on.

But if these all admit a vascular disease, they are far from admitting the same causal mechanism. We could divide into two classes the explanations given for a vascular origin of neuroretinitis, one considering the necessity of a real vascular lesion, which precedes the parenchymal process, the other admitting the existence of simple functional troubles, which are followed as a result, by parenchymal and vascular lesions.

v. Michel has admitted since 1899, a causal relationship between arteriosclerosis and retinitis. Vascular lesions (contraction, sinuosities, sclerosis) are usually found on ophthalmoscopic

examination. Recently, Wagener in 81 cases of neuroretinitis has seen 21 which had a normal kidney function (25 per cent.) while only six showed a normal arterial appearance. In fact, these vascular lesions are not seen in every case. Leber even, objected to v. Michel's theory, the absence of any correspondence between the retinal and the general arteriosclerosis, considering the first rather as a result of the disease than as a cause. He pointed out also, the lack of parallelism between the territories of distribution in the retina, of the sclerosis and the parenchymal lesions. In his report to the French Ophthalmological Congress in 1912, Rochon-Duvigneaud concluded against the arteriosclerotic theory. Recently, Kalt has published a new observation of neuroretinitis, in which he could not demonstrate any arterial lesion.

Such objections are avoided by those who admit that retinitis is due to a simple functional circulatory trouble, in connection with local or general hypertension. The importance of hypertension cannot easily be overlooked, because it appears to be one of the most frequent symptoms associated with neuroretinitis. Among our own observations we have met it 24 times in 29 cases. The same conclusion appears from larger statistics. (Wagener, Kollert.) Kollert in 85 cases found only three without hypertension, and eight where it was inconstant. He believes that in such cases, where one does not find actual hypertension, it must have previously existed at the moment of appearance of the disease. Spalding and Curtis, in a large number of cases, concluded that hypertension plays a great part in the genesis of retinitis diabetica. But how should hypertension produce the retinal appearances?

Leber thought that hypertension was due to a general peripheral arteriolar contraction and that the ischaemia resulting in the retinal territory was the cause of the lesions. Recently, Volhard admitted the same ischaemia as due to spasmodic vascular contraction (malignant hypertension). Such ischaemia would bring an insufficient oxygen supply, and consecutively nutritional troubles in the parenchymal tissues and the arterial walls, resulting in arteritis and retinitis.

But Volhard's spasmodic contractions are not usually seen. They are more frequently encountered in eclamptic neuroretinitis (Myllius). Most authors would not give to them a general importance. (Lo Cascio, Aschof, Schieck, Fahr). Hansen calls attention to the fact that sometimes an appearance of arterial contraction can be produced by retinal oedema, hiding the vessel. The majority of authors admit, therefore, that hypertension produces the neuroretinitis, through hyperaemia and stasis with the same result of anoxaemia and nutritional troubles (Schieck). Some authors are more reserved in admitting the causal action of hyper-

tension. (Chabanier, Charlin, Barlow.) In our own cases we could not find it always. Should we admit in such circumstances Kollert's view that hypertension had existed previously and had disappeared? Another possibility still remains, in admitting the action of local arterial hypertension. Bailliart tends to such an hypothesis. He recognizes (as does Morax) that local vascular trouble is at the basis of neuroretinitis, even when no arterial lesions can be demonstrated. This should primarily be a local hypertension which seems still higher than the value corresponding to the general arterial hypertension.

Anyhow the nature of the signs of neuroretinitis (haemorrhages, exudation), shows the direct participation of the circulatory troubles. The recent observations of De La Fontaine Vervey, tend to give a new support to those who admit the existence of arterial lesions. He made histological examinations of 23 eyes belonging to patients with atherosclerosis, among whom three had neuroretinitis. By means of special methods, he found a fine lipoid and protein infiltration of the walls of retinal arterioles, which would have escaped a more superficial examination. The interesting fact is that De La Fontaine Vervey did not find such lesions in retinal arteries, other than those belonging to the neuroretinitis cases. In all other cases the retinal arterioles were healthy, though the other arterioles (kidney, brain) were affected. The retinal arterioles would seem then, somewhat better protected against sclerosis and would yield only to stronger baneful influences. Such facts would explain the usual bad prognosis of neuroretinitis.

A new point of view is given by Koyanagi who points out the importance of choroidal vascular lesions. In an early case, he has found, by serial sections, that the branches of the central artery were healthy, whilst only the choroidal vessels showed sclerosis, corresponding to the territories of retinal lesions. This fact should explain why sometimes the retinal arteries are found healthy (as in Kalt's observation), if no serious examination of the choroidal vessels was made.

The next question to answer is, to what process is this circulatory trouble due? The French school in general, even admitting the rôle of vascular lesions, subordinates it to the pre-existing nephritis. Bailliart says: "All in retinitis albuminurica is subordinated to the kidney insufficiency; the circulatory trouble itself, if it is precocious is not primitive." In the same way, Leber considered hypertension as due to nephritis. Ambard and Yolande Levy seem to admit an association of renal insufficiency, with general hypertension. The German school, and, in France, Chabanier, and recently Terrien, consider the neuroretinitis independent of the kidney disease, having only an evolution parallel with it, and going on under the influence of common

causes. This was also the opinion that we had adopted in our previous paper. Kahler and Sallmann, suppose the existence of a special nervous centre of hypertension, which is susceptible to the different toxic substances of renal or other origin. Such a centre would be primarily influenced in the progression of the metabolic disturbances which characterize Bright's disease, and once the hypertension were produced, the local ocular or renal lesions would follow as a consequence. (Horniker, Schieck.)

Terrien and Renard in their recent paper, read at the Amsterdam Congress, conclude that "the ophthalmoscopic appearance of the so-called Bright's neuroretinitis seems to be produced by the association of multiple factors which produce a deep disturbance of the general state, manifested most frequently by hypertension and modification in the composition of the serum (lipoids, urea, etc.) These factors will produce in the eye vascular lesions, and perhaps due to those, or only in parallel with them, exudative changes. Excepting these, other special lesions will evolve at same time with the ocular lesions (especially kidney lesions)."

As pointed out previously our experience brings us to admit the existence of circulatory troubles in the genesis of Bright's neuroretinitis, independent of kidney insufficiency, and even of general hypertension. We think that the rare observations of the so-called retinitis pseudonephritica stellata, can bring a particular contribution for the understanding of these still undecided conditions. Having recently had the opportunity of seeing three such cases, we think it of interest to publish them, and to point out the suggestions which they may furnish. Our cases are as follows:—

Case I.—The wife of a peasant, aged 32 years, entered the clinic on March 10, 1929. No definite history of note before the actual disease began in June, 1928, with headaches coming in bouts of 20-60 minutes, which ended with vomiting, provoked sometimes by the patient herself, in order to put an end to her suffering. She had some drowsiness and more rarely faintings. Treatment had not brought any improvement. Last autumn she began to notice obscuration of sight and for the past two weeks she has become completely blind.

On examination she had no perception of light. The pupils were of equal width (6mm.) Tn., R. E. = 12 mm. L. E. = 15 mm. Ophthalmoscopic examination: R.E. The papilla seemed enlarged with oedematous pinkish appearance and greyish borders. The retina around the papilla was discoloured and speckled with numerous haemorrhages of varying size, which in places hid the vessels. L. E.—The borders of the papilla were indefinite, disappearing into a striated greyish retina, speckled with less numerous haemorrhages. The papilla was marbled with spots,

pink or pale. The vessels were veiled in its centre. The macula presented fine striations directed towards the papilla and reaching nearly to its border. (The image of a half-star.)

The knee-jerks were diminished. Babinski negative. The signs of Trousseau and Weiss were positive. Sensation was normal. The gait was difficult, zig-zag-like, with some tendency to staggering. C. S. F., tension 45-50 mm. (Claude.) Blood Wassermann, negative. Pandy, Nonne Appelt, slightly positive. The benzo-colloidal reaction negative. No lymphocytes. Albumin, 0·45 per cent. Urine: sp. gr., 1036. Chlorides 7 per cent. Urea 18 per cent. No albumin or sugar. Blood urea, 0·026 per cent. Cholesterol, 0·167 per cent. Calcium, 0·010 mgr. per cent. Chlorides, 0·620 per cent. Kidney function (Volhard) normal. General arterial tension 11/8. Retinal arterial tension 75/50. On one side, in the dorsal region, was a tumour, the size of a small orange, having a firm consistency and no very definite limits, fixed to the deep tissues. without adhering to the skin. A biopsy showed it to be situated under the muscular layer and to be very vascular. The excised fragment was pinkish-grey and showed on microscopic examination the structure of an adeno-carcinoma with groups of glandular acini, having one or two layers of vacuolated cells. In some places the constitution was frankly carcinomatous. No lesions could be detected in the genito-urinary tract, or in the mammae.

Case II.—A girl, aged 11 years, entered the clinic on November 21, 1929. The father died two weeks before the birth of the child, with pulmonary tuberculosis. The mother is healthy. She has another child, who is healthy, and has had no miscarriages. The patient has had measles and suffers frequently from colds and influenza. The general state was pretty good. Last summer she had an attack of malaria. On November 13 she observed an impairment of the sight of the left eye and this has since progressively diminished. No headaches. The body temperature has varied between 37·2° and 37·8° C.

On examination, R. V. = 1, L. V. = counts fingers at 30 cm. Ophthalmoscopic examination: L. E. The papilla was enlarged. pale yellowish with borders totally hidden. The vessels veiled at their emergence. The lower half of the papilla seemed swollen. Fine vascular branches appeared interrupted at the borders of the papilla, and then followed a sinuous course. On the external lower part of the macula some fine striation, having the disposition of a macular star, was present, with fine, yellowish, interrupted branches, reaching nearly to the border of the papilla. A small haemorrhage was situated between the star and the papilla. Nothing else to notice in the rest of the retina. The visual field showed a central scotoma.

General state:—Well grown, pale, presenting evident traces of

thoracic rickets. The left groin glands were swollen. On examination of the respiratory apparatus, the only thing to notice was the sign of D'Espine, which was frankly positive. The sinuses, frontal and maxillary were normal. Hypertrophic rhinitis on the left side. Hypertrophy of the Waldayer lymphoid circle. Persistence of the left temporary canine tooth, which has brought a vicious implantation of the permanent one: caries of the last left molar. The blood Wassermann reaction was negative. The intradermal reaction with tuberculin (1/10,000), negative. X-ray examination showed a marking of the hilar regions, with congestive reaction, especially on the right side. The parenchyma was clear. Laboratory examination: Urine: No albumin, no sugar, no casts. Ambard's ureosecretory constant 0·08. Blood examination: Neutrophile polymorphonuclear cells, 58 per cent., eosinophile cells, 4 per cent., mononuclear cells, 18 per cent., lymphocytes, 19 per cent., myelocytes, 1 per cent., transition forms, 3 per cent. Treatment: Injections with 0·50 urotropin daily, genostrychnine, phospharsyl, ultra-violet irradiation. Treatment of the nose and pharynx by specialist. On November 13 the papilla began to have a clearer delimitation, but still presented in the lower quarter the oedematous appearance. By December 8, the tumefaction had disappeared, the borders were clearer. The star was less pronounced, with fewer rays and more interrupted. The visual acuity increased to 1/3. The central scotoma was much reduced. The left papilla was pale with hazy edge and veiled vessels, which became apparent outside the disc. A group of white spots with sharp outline and the size of a pin's head, or smaller was seen near the inferior part of the papilla. Two white nodules were present near the superior edge. The macula half star still persisted. Small groups of pale points spread in the inferior part of the peripapillary retina.

Case III.—A male peasant, aged 41 years, entered the clinic on January 13, 1929, for diminution of the sight of the left eye, observed during the past 12 days. He is married, has healthy children, does not smoke or drink. Has had diphtheria in childhood.

On examination, R. V. = 0·4, L. V. counts fingers at 10 cm. Ophthalmoscopic examination: R. E. nothing abnormal. L. E. The borders of the papilla and the peripapillary retina were striated and greyish. The centre of the papilla was more coloured, the emergence of the vessels totally veiled, the vascular branches appeared at the border of the disc. The two superior branches disappeared under a white spot, of medium size, situated near the superior border of the papilla. From that spot the vessels continued distended and sinuous. In the inferior part, there was another white spot, covering a vessel which emerged from the border of the

papilla. Well formed macular star, with numerous pale, long, branches. The branches were less numerous on the external side. The visual field presented a slight narrowing to the right. To the left, the patient recognized the colours only at the fixation point. The retinal arterial tension 75/25 mm. The serodiagnostic blood Wassermann reaction, negative. General arterial tension 15/7. No cardiac or pulse abnormality. Azothaemia, 0·042, cholesterol, 0·10 per cent. Urine, no sugar, no albumin, urea 2·4 gr. per cent. uric acid, 0·026 per cent., kidney permeability (Volhardt) normal C. S. F., tension 47 mm. (Claude.) Lymphocytes 4·5 per c.mm. Nonne Appelt, negative, albumin, 0·022 per cent.

The patient was treated with strychnine. Subsequent course: January 27, 1929, L. V., 0·2. Visual field for white from 40° to 30° and for colours from 30° to 15°. He left the clinic and returned on March 20. R.V.=0·4, L.V.=0·3. We then found retinal lesions on the right side as well. The papilla was striated on the temporal side with borders more red and less regular than in the rest. The papillary and macular region were occupied by small white bright spots. In the left eye the appearance was unchanged. Visual field normal in the right, narrowed in the left eye. He continues to have no other general trouble except the retinitis.

The patient from the first observation had signs of intracranial hypertension and a tumour adherent to the ribs, of adenocarcinomatous constitution, very probably of metastatic origin. It is therefore possible that the intracranial hypertension might have been due to a cerebral metastasis and we could place this case in the same category as those published by Colrat, where a retinitis pseudonephritica had appeared during the evolution of cerebral tumours. Before Colrat, Paton and Holmes have spoken of a macular form appearing sometimes with papillary tumour oedema. On the other hand, Pick has described as "retinitis cachecticorum," lesions appearing in the retinae of cachectic patients, especially carcinomatous, and showing haemorrhages and white spots. In his cases the anaemia was extreme.

L. Werner also has observed a girl, aged 24 years, suffering from chlorosis, who had unilateral neuroretinitis with papillary and peripapillary oedema and macular star. Another such case was published by Augstein.

Case Π could be put down to a local tuberculosis (although the negative intradermal reaction would be against this diagnosis). Recently, Kaminskij saw a case of neuroretinitis, with unilateral star, no renal lesions, but coexisting with pulmonary tuberculosis, which was proved by tuberculin treatment. In the observations published by Kleiber, the papillary and peripapillary oedema and haemorrhages were coincident with a choroidal focus and with a

typical star. Kidneys and arterial tension were normal and only a tuberculin reaction justified a diagnosis of a tuberculous process. Presence of stars in periphlebitis retinae tuberculosa has been observed by Sydler Huguenin and Schieck.

The cases of neuroretinitis stellata pseudonephritica, appearing after an infectious disease are still more frequent. Epidemics of influenza have given a specially large number of cases. Danco quotes the observations of Leber, Hartridge-Gross and Maclakof, and gives the history of one case of his own, with neuritis, exudates and macular star, coinciding with normal kidney function. The arterial tension was only 120 mm. but a slight rachidian hypertension thought to be due to meningitis serosa was present. This case was recently seen again by Hessberg (discussion in Stewens's paper) who found optic atrophy developing. We could compare it with our third observation, where we have found also intrarachidian hypertension.

Stewens has published an observation of bilateral neuroretinitis with macular star appearing five days after measles, with a normal kidney function, but a transient hypertension 115/60 (at the age of 6 years) becoming after three days 95/60. Various retinal lesions have in fact been observed, following measles.

R. Onfray has had a case of unilateral neuroretinitis with normal kidney function which was improved by antiluetic treatment.

Junius, in a series of four observations, which he collects under the name of " retinitis exudativa juvenilis," has one with the presence of bilateral macular star. This was a woman who was under observation from the age of 14 years to 40, when she died, with an aortic aneurysm. Although the Wassermann reaction had been negative, the author questions if the aetiological factor was not hereditary syphilis. The kidney and the arterial tension were normal. The ocular lesions later retrogressed.

Pseudonephritic neuroretinitis with macular star has been described also by Coppez (unilateral lesion connected perhaps with bad teeth); by Pascheff (no precise aetiology); by Schieck (Korsakow's syndrome). Nitsch had a case appearing after a cranial trauma, with hemiparesis and aphasia.

Magitot has seen a case where typical retinal lesions appeared in immediate connection with slight corneal trauma. The sight diminished immediately. Examination made 17 days after the accident showed the characteristic exudates, papillary oedema and star, coinciding with a normal general state. The signs disappeared totally after six months. Of course, it is not certain that the ocular lesions were not there before the trauma, which gave rise to an ophthalmoscopic examination. Magitot inclines to attribute the cause to the trauma.

Notwithstanding the diversity of aetiological factors, all these

observations present a common ophthalmoscopic image, and evolution with a relatively mild prognosis, absolute independence of any renal lesion and of general arterial disease. At the same time, the presence of a macular star similar to that found in the course of a Bright's neuroretinitis inclined Leber to create a special entity, the retinitis pseudo-albuminurica stellata. In fact, the presence of the macular star in the course of Bright's neuroretinitis is very inconstant. We have met it three times in our total of 35 observed cases. On the contrary, as Schieck points out, the star is found much more frequently in the pseudonephritic forms, and is due to the particular disposition imposed on the exudates by the radiate architecture of the perifoveal region.

On the other hand, such cases if they have the local symptomatic appearance of a Bright's neuroretinitis, do not have any nephritis or hypertension. Should we admit then that the same ophthalmoscopic image is due in both cases to two different mechanisms? That is why Schieck doubts if in the cases of pseudonephritic neuroretinitis, which have been previously published, and where no mention about arterial tension was made, this last could not have been the causal factor. But our present observations and some others (Kleiber, Junius, Danco), where the blood pressure was measured, showed it to be normal.

The neuroretinitis pseudonephritica stellata becomes still more interesting if we consider it (as Junius does), as a transition form between typical nephritic neuroretinitis and the different forms of central exudative retinitis which show a more variable fundus picture.

Transition towards the typical Bright's forms could be found in some of the cases of Pick (neuroretinitis in cachectics). In these, besides the typical ocular symptoms, the clinical appearance suggested the existence of a kidney lesion, but the necropsy showed the total absence of such lesions and only a simple oedematous congestion. Just the same occurred in a case of Marx and Hellmuth. The evidence of kidney participation comprised albuminuria and casts, appearing only after physical exertion, with an insufficient response to the water test. The author attributes these signs not to the nephritis but to a general disease of the endotheliocapillary system.

In Bakker's case, a unilateral retinitis with oedema, haemorrhages and star, was co-incident with a normal circulatory system, and only traces of albumen in the urine. In this case too one should think rather of direct action of influenza toxin on the retina. Still more doubtful was the nephritis in a case of Farnarier, which besides the typical ocular signs had an azothemia of 0·3 only in one determination and was considered by the author as transient.

Another group in this transition series is formed by the cases where we have the picture of neuroretinitis but without any star. Such is Pick's fifth case where the condition had appeared in a woman with grave carcinomatous anaemia. Nageldinger has observed a bilateral neuroretinitis with cyclitis, following on influenza. Well studied is also the case of Menacho where a typical ocular picture was co-incident with normal blood, urine and arterial tension, and a negative Wassermann reaction.

Then at the other extremity of the series we can consider the four observations of Junius, which he calls " retinitis exudativa juvenilis." One case had a macular star and was at first considered as the pseudonephritic form of Leber. All three others had a more variable appearance, characterized in general by exudative spots of different size and various distribution, accompanied by haemorrhages and papillary lesions. Junius compares his observations with those of pseudonephritic neuroretinitis of Augstein and Danco, which he considers to be of the same nature. "Each of these observations gives a particular characteristic to the picture of the disease. But all have in common a related clinical picture, which, although variable in its appearance, is due to vascular circulatory lesions in the small vessels, which have an essential importance for the life of the macula."

Such exudative changes have been observed also in cases of the neuroretinitis of Bright's disease. So we could consider the observation of De Logu where coincidently with renal trouble, urinary casts, aortic reflux and cardiac hypertrophy, there was a picture of Bright's neuroretinitis in one eye and one of retinitis circinata, with white spots disposed as a horse-shoe around the macula, in the other eye. There were some haemorrhages in this eye and the spots were situated beneath the vessels.

We could consider Horniker's eight cases in the same group. He collected these under the name of " retinitis centralis of vasomotor origin." In them the ophthalmoscopic image presented haemorrhages, oedema, and small whitish foci, and at the same time a contraction of the fine macular vessels attributed to a capillary spasm.

In conclusion all these forms have in common the presence of white spots of different sizes, localized in the posterior pole and accompanying the papillary lesions, the haemorrhages and sometimes, even choroidal lesions. Another characteristic fact is the tendency to resorption, which in fact belongs in certain conditions also to the spots of the typical neuroretinitis of Bright's disease.

Should we consider the exudates of Bright's neuroretinitis as being of a different nature? We distinguish in this disease two kinds of white spots. The one is due to a local degenerative gangliform tumefaction of the nerve fibres. Such lesions have

been met with not only in the retina, but also in the posterior roots of the spinal cord, in the course of various affections. (Roth, Minich.) Such are retinitis pigmentosa, retinitis cachectichorum, lues, sepsis and brain diseases, for the retina; pernicious and bothriocephalus anaemia for the cord. Minich admits that blood changes and spinal changes have the same origin, from the production of toxic substances. Roth considers the retinal varicose hypertrophy of nerve fibres as the expression of an inflammatory process.

Other white spots are of exudative origin and the majority of authors consider them of vascular origin. We have seen that the tendency is to attribute to arterial hypertension the predominant rôle in their production.

In quite a number of the cases previously recorded a simple local vascular process of toxic origin could be admitted. Such are the cases in the course of carcinomatous anaemia. One could not speak in these instances of hypertension. On the other hand fundus lesions have been observed in the course of essential pernicious anaemia (described more recently by Fileti), but they do not have the characteristic exudative form as in Pick cases. For an explanation of the latter, we should be inclined to admit, in accordance with Pick, a local toxic action due to the progress of the carcinomatous process. Perhaps in the cases with luetic and tuberculous origin the same explanation might be true.

Should we, as Bailliart does, accord more importance to the local arterial tension and explain the genesis of neuroretinitis on the basis of purely functional, circulatory troubles? Bailliart has found in Bright's neuroretinitis the retinal arterial tension increased in a greater proportion, than we should expect from the general hypertension. One could easily imagine cases where only the retinal tension should increase, with a normal general tension. Such could be the genesis of neuroretinitis in the course of cerebral tumours and perhaps of those of periphlebitis retinae tuberculosa. Intrarachidian hypertension could play a part also in the genesis of the local circulatory trouble. Danco's patient showed it, perhaps the case of Nitsch (which followed cerebral trauma) belongs to the same class. Two of our three patients had hypertension of the cerebro-spinal fluid but the retinal arterial tension at the moment of our examination was respectively 75/50, and 75/25.

There is, however, no necessity to admit local hypertension as a rule. Foster Moore and Duke-Elder,* think that the retinal capillaries, if perfectly healthy, should resist variations of

* W. STEWART DUKE-ELDER.—Recent Advances in Ophthalmology. Second Edition, p. 240. London: J. & A. Churchill. 1929.

pressure without any leakage. "The very essence of retinal haemorrhages is probably capillary dysfunction rather than high blood pressure itself . . . . Haemorrhages, together with exudates, are the result of injurious processes affecting their walls, or are an expression of defective circulation through them so that their endothelium is impaired." According to these authors the primary cause remains thus only an endothelial impairment, be it due to circulating deleterious toxins, or when arteriosclerosis complicates the picture, to an insufficient blood supply, adding its effect to the suffering endothelium.

There is, however, a particular class of case in which we must admit that the beginning of the lesions is the result of a simple mechanical defect of the circulation. These are the cases of pseudo-albuminuric retinitis appearing during the evolution of an intracranial tumour. We cannot discuss here the very complex problem of papillary stasis. Leber admitted once an inflammatory pathogenesis. Some authors saw in it vasomotor troubles. Recently Magitot considers papillary oedema as a result of a defect in the functions of the capillary walls. But the majority of authors will admit to-day a mechanical pathogenesis as a result of an increased intracranial pressure. The authors differ in their explanation as how the intracranial pressure will produce a papillary stasis. Paton and Holmes have admitted that intracranial pressure transmitted to the optic nerve sheath produces simultaneously the increase of pressure in the central vein and a stasis in the lymphatic circulation. Only by the association of these two causes should a papillary stasis result. Schieck, Behr Lewinsohn, Abelsdorf, although admitting somewhat different mechanisms accept in fact the compression lymph stagnation. This theory can explain at best the papillary oedema associated with high intracranial pressure (tumours, abscesses), eventually serous meningitis, although in the latter, an inflammatory process could perhaps play a part. Some will admit that any papillary oedema, even that of Bright's neuroretinitis is always due to a high intracranial pressure and produced in the same way. Paton and Holmes distinguish sharply between the mechanical lesions due to tumour hypertension and the inflammatory process seen in nephritis or arteriosclerosis. Behr speaks in such cases of an active lymph stasis due to a real transudation of lymph through disturbed capillary walls. All admit that during the evolution of the disease, inflammatory reactions of the tissues add themselves to the previous mechanical distension of the tumour papillary stasis.

Now, should we admit that the pathogenesis of the clinical aspects which simulate a Bright's neuroretinitis is different in the case of cerebral tumours from what it is in the other cases?

We tend to accept the mechanical " primum movens " in the production of tumour stasis, but as Schieck has pointed out, and as Volhard has admitted, in his theory of capillary spasm and retinal ischaemia, such circulatory trouble could not last without bringing local nutritional troubles and consecutive lesions to impose themselves on the simple initial picture.

In what concerns the exudative forms of retinitis (Bright's or pseudo-Bright's), we tend to admit the so-called inflammatory theory (taking the word inflammation in a broad sense) as discussed previously in this paper. In the case of tumour neuroretinitis the " primum movens " might be a mechanical one. But no matter what the " primum movens " might be, the so-called inflammatory process, the reaction on the part of vessels and retina must appear and contribute to the clinical picture of neuroretinitis.

Paton and Holmes themselves point out that in some cases a clinical differentiation between these two syndromes becomes impossible. Thus, the local lesions constituting the clinical syndrome can have their origin in general, metabolic, toxic disturbances (case of Brightism) or at other times, in the special conditions which follow local metabolic troubles, resulting from the mechanical action of the tumour stasis. But their evolution remains related just as their clinical pictures which can sometimes be confused.

The theories which tend to explain the production of the retinal lesions must consider also the fact of their localization at the posterior pole of the globe. Schieck explains this on a functional basis. In view of the fact that we do not find in the central portions of the retina a greater ramification of vessels than in the rest of it, although the physiological activity is much greater, one should admit that even in normal conditions, the walls of the arterioles which are situated in that portion must satisfy such increased activity by an increase of permeability. Immediately the equilibrium of pressure between the content of the vessels and their exterior is changed as a result of hypertension, this will be the territory where the first exudation will take place.

Schieck quotes the observation of Bartels, where the neuroretinitis appeared unilaterally while the other eye, aphakic for some years and amblyopic, remained without signs of retinitis. So should we explain also the observation of Greeff ,where the signs of neuroretinitis disappeared when an attack of glaucoma occurred.

Koyanagi sees the central localization of the lesions, as a localization in the territory corresponding to the choroidal lesions, in the portion where the capillary net, which is important for the nutrition of the retina, is especially close-meshed.

In fact, the analysis of the cases related in this paper brings us to the same conclusions as others have found, that for the cause of the characteristic white spots and oedema, there is not of necessity any nephritis, or even a general hypertension. But admitting the rôle of the local circulatory trouble, we are still far from a full explanation of the pathogenesis. Bailliart questions why from all the retinal transudations and haemorrhages (the oedema of traumatic commotio for instance) which are so easily resorbed, only those of Bright's neuroretinitis (or we should say all retinal exudative forms), give persisting deposits. Admitting the rôle and origin of fibrin, as it was described by R. Duvigneaud and Mawas, we must still admit that a special element found in the serum gives to the exudates a special character which makes them lasting.

Even this again has brought us to admit the existence of special pathological elements present in the blood. But whilst for Bailliart these are of renal origin, we believe that various products will play the same rôle among which eventually we could count those of nephritic origin. It is worth while to note that Kollert found in the cases with neuroretinitis and malignant hypertension, an increase in blood fibrinogen, and of the sedimentation of erythrocytes.

How should we explain that among the various exudative phenomena described, the picture of Bright's neuroretinitis is so characteristically constant? Probably this is due to the constant presence of the same metabolic troubles, which are at the basis of the whole syndrome of retinitis and nephritis and have a constant and characteristic way of affecting the retina and the vessels.

In fact, the pictures of the different neuroretinitides are much alike. Some authors have tried to see sufficient differences to establish an aetiological classification based on ophthalmoscopic appearance. Especially have arteriosclerotic forms been isolated from the usual toxic forms. (Foster Moore.) But such distinctions are still uncertain, and we have not yet decided if the retinitis of diabetes has really its individuality, and a true constant characteristic picture, as opposed to the albuminuric forms.

We should rather admit with Duke-Elder*: "In a tissue so highly differentiated as the retina, it is only to be expected that different toxins acting upon it would produce pathological alterations of a closely related type, the nature of which would be determined rather by the structure itself than by the causal agent. At the same time it is reasonable to suppose that the deficient circulation in arteriosclerosis may tend to introduce differentiating peculiarities, and that in diabetes the upset in the water balance . . . should be able to influence in a similar manner the backward

* W. STEWART DUKE-ELDER.—*Recent Advances in Ophthalmology*. Second Edition, p. 244. London: J. & A. Churchill. 1929.

continuation of the posterior epithelial layer in the retina, endowing on occasion the manifestations of the disease in this tissue with clinical characteristics of its own." In the same manner we could admit that the particular conditions presiding over the production of Bright's disease would give the particularities of the clinical picture.

The existence of different toxic processes all of which cause a retinal lesion (among which are included those due to Brightism), would explain the variable prognosis in different cases. We know that the prognosis of true Bright's neuroretinitis was considered very grave, *quo ad vitam*. On the contrary the pseudonephritic forms do not menace life and their local evolution is generally favourable. Latterly, even the prognosis of nephritic neuroretinitis seems less grave than it was considered before.

Among his 85 patients, Kollert found seven with a duration of over 2 years. One of these had a history of 10 years, and the others 8, 7, 5 and 4 years respectively. He thinks that the bad prognosis is connected with a permanent hypertension, and if this tends to return to normal in the evolution of the disease, the prognosis becomes better. This shows that the prognosis is a function of the intensity of the general trouble and that even in nephritis this is not always so bad as to lead to early death.

We could not, unfortunately, follow up all the 35 cases of neuroretinitis over a long period. We have, however, a case of 7 years' duration in which the retinal signs have disappeared and the visual acuity has increased to 1. Another patient is also alive 8 years after the diagnosis of neuroretinitis was made, notwithstanding marked hypertension (24/17 at the time of the first examination), which has continued till to-day. This might show that even hypertension alone is not always a true index for the prognosis.

The relatively severe prognosis of Bright's neuroretinitis shows at any rate the seriousness of the general trouble in Brightism. If this happens to recede, or to remain unprogressive the lesions of neuroretinitis themselves do not seem to be more persistent than in the other exudative forms. The resorption of the eclamptic forms, the periodicity of those in pregnancy, even the close observation of the other cases, show that there is the same tendency to heal. Brightism remains a very serious disease and even if we can treat it better to-day and give it a milder prognosis, it will be still far from the benignity of the other causes of neuroretinitis, and the appearance of this, in the course of nephritis remains a proof that the disease is progressing in a menacing way, and that the patient is in danger *quo ad vitam*.

Summary.—Retinitis pseudo-albuminurica stellata (among which we class our three cases given in this paper) occupies an

intermediary position, as a transition between the typical Brightic forms and the different exudative ones, as described by Junius and Horniker. Their study gives us a better understanding of the pathogenesis of Bright's neuroretinitis, suggesting to us a similar mechanism in the production of the lesions.

The different aetiological factors acting on the retina (acute or chronic infectious diseases, cranial traumata, cerebral tumours, serous meningitis) determine (probably through a vascular mechanism) their related pictures, which represent somewhat the special kind of reaction of the retina, and differ in details according to the different cases and aetiologies. At the origin of the pseudo-Brightic retinitis, due to intra-cranial tumour, there seems to be a mechanical lymph stasis, causing the papillary oedema and through it metabolic troubles. At the origin of the pseudo-Brightic, infections, etc., retinitis the local troubles seem under the dependence of general metabolic processes. All follow thereafter a similar evolution and produce related fundus pictures.

The neuroretinitis of nephritics is not connected by any causal link with the kidney lesions and therefore the only justified name should be that of Bright's neuroretinitis (understanding Brightism as a general disease, with renal, ocular and general manifestations.)

From the facts discussed here, we incline to admit that the production of the neuroretinitis is independent of arterial hypertension, which like the renal lesions, is only a symptom in evolution under the influence of the same causes.

The difference in evolution and prognosis between the Brightic forms and the so-called pseudonephritic is due to their different aetiology and to the intensity with which this affects the general state. This explains the benignity of those due to local processes and on the contrary the gravity of those where the whole organism is participating (hypertension, Brightism).

The characteristic clinical picture of Bright's neuroretinitis, shows that the special metabolic trouble, which forms its basis, has in its nature intensity and distribution of vascular action, particular and constant characteristics.

We think it might be of interest to group in a general table the observations we have discussed.

I.—Observations of neuroretinitis pseudo-albuminurica stellata which we have found in the literature.

Author	No. of cases	Aetiology	Ocular signs	Kidney function mentioned	Blood pressure mentioned
Colrat - - -	2	Cerebral tumour -	Papillary oedema, haemorrhages, star	Normal	—
Magitot - - -	1	Ocular traumatism -	Unilateral. Papillary and peripapillary oedema, exudates, star	Normal	—
Schieck - - -	2	Periphlebitis retinae tuberculosa	Macular stars. Papillary stasis in one case, papillary focus of periphlebitis in the other	Normal	—
Sydler-Huguenin -	1	Tuberculosis of the optic nerve	Neuroretinitis with star - - -	—	—
Kleiber - - -	1	Uncertain. Tuberculin reaction positive	Unilateral. Papillary and peripapillary oedema, haemorrh., star, choroidal focus	Normal	Normal
Kaminskij - -	1	Pulmonary tuberculosis	Unilateral Albuminuric neuroretinitis, star	Normal	—
Junius - - -	1	Hereditary syphilis?	Bilateral. Veiled papilla, exudates, star. No haemorrhages Contracted vessels	Normal	Normal
Werner - - -	1	Chlorosis ? - -	Papillary oedema. Star appearing five days later on the edge of a big macular exudate	—	—
Stewens - - -	1	Measles - - -	Bilateral. Typical appearance of Bright's neuroretinitis with star	Normal	Transitory hypertension 115/60 becoming 95/60 in 3 days (6 years of age)
Vauce - - -	1	Scarlet fever - -	—	—	—
Leber - - -	1	Meningitis - -	—	—	—
Vossius - - -	1	Facial erysipelas -	—	—	—
Hartridge-Gross -	1	Influenza - -	—	—	—
Maklakoff - -	1	Influenza - -	—	—	—
Danco - - -	1	Influenza - -	Bilateral. Neuritis optica, oedema, exudates, star	—	—
Neubner - - -	2	? - - -	—	—	—
Coppez - - -	1	Dental origin ? -	Unilateral. Exudates, haemorrhages, star	No albumin in urine	—
Pascheff - - -	1	? - - -	Retinitis, star - - - - -	No nephritis	—
Chevallereau - -	1	Syphilis ? - -	Neuroretinitis, star. Double paralysis of oculomotorius externus	—	—

II.—Aspects of pseudonephritic retinitis without star and related exudative aspects.

Author	No of cases	Aetiology.	Ocular signs	Kidney function mentioned	Blood pressure mentioned
Nitsch	1	Grave cranial trauma, hemiparesis and aphasia	Picture of Bright's neuroretinitis	Normal	—
R. Onfray	1	Hereditary syphilis	Unilateral. Picture of Bright's neuroretinitis	No albumin in urine	—
Pick (5th case)	1	Grave anaemia in gastric carcinoma	Typical appearance of Bright's neuroretinitis	—	—
Zimmermann	1	Influenza	Bilateral. White rays and spots around the papilla	—	—
Nageldinger	1	Influenza	Bilateral. Picture of neuroretinitis with cyclitis	—	—
Menacho	1	?	Bilateral. Retinal oedema, haemorrhages	Normal	Normal
Pick	8	Grave anaemia in cancer of the alimentary canal	White or greyish spots of variable size. Haemorrhage. Sometimes papillary oedema. 3 cases with unilateral lesions	--	—
Pick	2	Grave anaemia in profuse haemorrhages	Same appearance. 1 case unilateral	—	—
Pick	6	Anaemia following other diseases	Same appearance. 1 case unilateral	—	—
Terson	1	Dental infection	Papillary oedema, haemorrhages	—	—
Junius	3	1st. Menstrual trouble?	Diffuse oedema, exudate focus	Normal	—
		2nd. Unknown toxemia	Unilateral. Exudate focus in macula, haemorrhage, white spots	Normal	--
		3rd. Ophthal. migraine	Unilateral. Macular exudate w. local am. retinae	Normal	—
Horniker	8	Vasoneurotic diathesis	Characteristic changes. Oedema, haemorrhage, fine white spots, vascular contraction	—	—
Cases with doubtful kidney impairment and doubtful nephritic origin.					
Farnarier	1	?	Unilateral. Papillary oedema, white spots, haemorrhages	Transitory 0·39 azothem.	Normal
Bakker	1	Influenza	Papillary oedema, star, haemorrhages	Traces of albumin	Normal

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ULTRA-VIOLET RAY TREATMENT OF TRACHOMA

BY

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OF the making of treatments for trachoma there is no end. Most of them are a weariness of the flesh and many of them are positively brutal.

Trachoma is a disease which already causes much suffering and discomfort to the patient. It may be asked why add to the patient's suffering? Is there a treatment which is pleasant, rapid in its effects, and definitely curative? There is.

In July, 1928, I published a note in the *China Medical Journal* giving in detail a technique for the treatment of trachoma by the open tungsten arc-lamp. (Here let me emphasize that treatment to be effective must be by the tungsten arc-lamp, *not* by the mercury vapour lamp, which is of much less value for the purpose and liable to vary in its output of ultra-violet rays while emitting an excessive amount of the far ultra-violet rays when active; so that one cannot use such a lamp close up without undesirable results. Neither is the carbon arc-lamp of use, for it only emits 5 per cent. of ultra-violet rays.)

The patient is seated opposite the lamp at a distance of three feet and told to close the eyelids gently as if asleep. One drop of a 1 per cent. solution of adrenaline is instilled into each conjunctival sac in order to denude the parts of blood as much as possible, and so remove all impediment to the passage of the rays. Each eye is irradiated for a period of two to three minutes according to the severity of the case.

Ultra-violet rays are said by the physicists not to penetrate beyond the stratum corneum. This is *in fact* quite untrue, as any one can prove by their potency for sterilizing the conjunctival sac.

In cases of trachoma accompanied by super-added pneumococcal, streptococcal, staphylococcal, Koch-Weeks and Morax-Axenfeld infections one can observe with extreme satisfaction the rapidity with which these infections clear up under ultra-violet ray treatment, as also does any chronic blepharitis or other suppurative condition of the lids. I use a 6-10 ampère lamp and treatment is given at 3-day intervals.

In Hong-Kong I found that patients were so gratified by the pleasant results of the treatment that they frequently anticipated their subsequent treatment and begged to be treated more frequently, which is of course an unwise procedure. It is highly improbable,

one may add, that patients would express such gratification if the rays did not penetrate further than the stratum corneum. As a matter of fact the rays do penetrate to the conjunctival sac, as any ophthalmologist can prove to his own satisfaction if he cares to make use of the tungsten arc-lamp for sterilization of the conjunctival sac before operation.

It is the long wave lengths that are the most effective for this purpose and it is likewise these long wave lengths that are more penetrative, and these are present in high percentage in the tungsten arc-lamp in addition to the short wave lengths. I look upon ultra-violet radiation as the most useful method of sterilizing the conjunctival sac.

I treated over 200 cases of trachoma successfully in private in Hong-Kong, there being no ultra-violet ray installation available at the hospital. The time of both doctor and patient is saved and the gratitude of patients after one or two treatments is a pleasure to observe. Most of the patients stated that they had had no treatment which caused them greater relief and comfort soon after the first application.

I am of the opinion that trachoma could be rapidly eradicated from a community or school if an intensive campaign by team work were established, using tungsten arc-lamps, under expert supervision.

ABSTRACTS

I.—PATHOLOGY

(1) **Blegvad, O.** (Copenhagen).—**Boeck's sarcoid of the conjunctiva.** (**Boeck's Sarcoid der Conjunctiva**). *Acta Ophthal.*, Vol. IX, p. 180, 1931.

(1) The condition known as Boeck's sarcoid of the skin was described in 1899 under the term of multiple benign sarcoid tumours of the skin. It is now recognized as a general affection. In the skin, nodules of varying size develop: from a few millimetres to several inches in diameter. If these are pressed upon by a glass, apple jelly-like nodules, similar to those observed in lupus, but smaller, are seen. The sites of election are the face, back, neck and extensor surface of the arms. Microscopically collections of epithelioid cells with an occasional giant-cell are seen: small round cell infiltration, so characteristic of lupus, is very sparse. In the smaller of the long bones, X-rays show up

typical cyst-like spaces. Mucous surfaces (tongue, nose and tonsils) may become involved, as also lymphatic nodes and parenchymatous organs. Microscopically the changes are similar to those in the skin.

Ocular complications of this general condition have been reported, especially iritis and irido-cyclitis. **Blegvad** has collected from the literature five cases of conjunctival involvement and reports three more observed by himself. Only two of the five reported cases are held by the author to have been cases of Boeck's sarcoid of the skin, and on the basis of the five remaining cases he constructs a general picture of conjunctival involvement in this disease. The nodules affecting the conjunctiva may vary in size and number, from a few discrete dots to a completely studded tarsal conjunctival surface. The nodules are mainly clear, yellowish follicles. There is on scarring, though in an old-standing case a tendency to symblepharon has been seen. The pre-auricular glands are not involved. The nodules show no tendency to involve the cornea; they have a chronic course which is unaffected by the administration of light and arsenic, remedies which affect the skin lesion favourably.

The nature of the affection is unknown. Its relationship to mild tuberculosis and to Mikulicz' disease is discussed.

ARNOLD SORSBY.

(2) **Daniels, B. (Hamburg). — Sympathetic ophthalmitis in choroidal sarcoma. (Zur Frage der sympathetischen Ophthalmie bei Aderhautsarkomen).** *Zeitschr. f. Augenheilk.*, Vol. LXXIV, p. 176, 1931.

(2) **Daniels** reports a case of iridocyclitis with secondary glaucoma in the eye of a patient in whom the other eye was the seat of a choroidal sarcoma. The eye with the tumour showed a detachment for which no operation was performed, and when ultimately the eye was removed, the neoplasm was seen to be still confined within the sclerotic. The literature on the subject of sympathetic ophthalmitis in intra-ocular new growths is critically reviewed. The author holds that the pigment cells of the tumour have some connection with the sympathetic inflammation, all reported cases having occurred in pigmented sarcomata. Another factor is necrosis of the tumour cells: for it is only when these break down that the pigment can be liberated. The reasons why sympathetic ophthalmitis is rare in tumours are the early removal of such eyes and the infrequent decomposition of the cell mass.

ARNOLD SORSBY.

(3) **Seefelder, R. (Innsbruck).—Glioma of the optic nerve. (Beiträge zur den Gliomen des Sehnerven).** *Wien. klin. Wochenschr.*, Vol. XLIV., p. 838, 1931.

(3) In 11½ years, in a series of about 60,000 cases, two cases of glioma of the optic nerve were seen in Seefelder's clinic. These cases were reported upon by Hidano in 1925 (*Zeitschr. f. Augenheilk.*, Vol. LVII, p. 31). **Seefelder** now reports on their late history.

In the first case, operated upon in 1920, the tumour was only partially removed through a Krönlein approach. The child is in good health and the eye, though showing some evidence of degeneration and prone to be irritable, is in a satisfactory state.

The second case, a child of eight months, had a Krönlein operation in 1924. Practically the whole of the orbital part of the optic nerve was removed, but two years later the child was brought back with a recurrence of the exophthalmos for which a further operation was performed with but little effect. A year later the child was re-admitted and found to have hemiparesis due to frontal cortex tumour. Nothing was done and the child is still alive, though very cachectic, the orbit being filled with neoplastic tissue.

The severe course of the second case is contrary to general experience which regards glioma of the nerve as an innocent growth. In view of the preponderance of ocular signs and the comparatively late involvement of the brain, Seefelder holds that the growth originated in the orbital part of the nerve.

In contrast to this he reports a case of a blind boy, aged 9 years, in whom autopsy revealed glioma beginning in the intracranial part of the nerve. In such cases the ocular symptoms are limited to optic atrophy and blindness; there is no exophthalmos and no disturbance in motility. These cases are exceptionally rare.

ARNOLD SORSBY.

(4) **Lindberg, J. (Helsingfors).—Three cases of glioma of the optic nerve. (Drei Fälle von Gliom des Sehnerven).** *Acta Ophthal.*, Vol. IX, p. 200, 1931.

(4) Primary glioma of the optic nerve must be distinguished from secondary invasion of the nerve by extension of a growth from the globe or brain. In reporting three cases of this rare tumour, **Lindberg** points out the extreme variation in structure shown by different parts of the same tumour. The classification of these tumours, besides the obvious one of tumours arising from the sheaths and those from the nerve tissue, is discussed. Besides the classical clinical signs of increasing exophthalmos,

good ocular motility and the propulsion of the eye straight forward, the author stresses easy dislocation of the globe from between the lids.

One of the author's cases received X-ray treatment to the maximum dose without any beneficial result and ultimately came to operation, Krönlein's operation. The patient died from meningitis. The author pleads for the safer orbital explorations after the manner of Knapp or Lagrange, especially when the eye is damaged.

ARNOLD SORSBY.

(5) **Bulac, C. O. (Baku).—The morphology of the neuroglia in the optic nerve and retina as revealed by the methods of the Spanish school [Ramon y Cajal] (Zur Morphologie der Neuroglia im Nervus opticus und der Retina nach Methoden der spanischen Schule. [Ramon y Cajal]).** *Zeitschr. f. Augenheilk.*, Vol. LXXIV., p. 248, 1931.

(5) **Bulac's** paper is well illustrated. His material was mainly oxen eyes, for he found that human, goat, sheep and rabbits eyes had the same structure. On special points such as the types of astrocytes there were some differences and these were specially studied. He concludes that Del Rio Hortega's technique is the most satisfactory for the study of neuroglia, completely supersedes the methods of other Spanish observers, and gives most promise that with modifications it will solve the outstanding problems in this connection. One such modification is suggested by the author, whose work in the main confirms the findings of Marchesani. By staining the retina with gold sublimate after the method of Ramon y Cajal the author could demonstrate astrocytes in the tissue; staining after Hortega shows up Müllerian supporting fibres.

ARNOLD SORSBY.

(6) **Ehlers, H. and Okkels, H. (Copenhagen).—Mixed tumour of the lacrymal gland. (Tumeur mixte de la glande lacrymale).** *Acta Ophthal.*, Vol. IX, p. 68, 1931.

(6) **Ehlers** and **Okkels** report a case of their own and review the literature on mixed tumours of the lacrymal gland. They conclude there are two types of growth, one encapsuled and innocent, the other lowly malignant, with a tendency to break through the capsule and burrow into the orbit. These types are apart from the frankly malignant tumours.

From the point of view of treatment, these tumours must be regarded as malignant. They are refractory to radiotherapy and

owing to the possibility that they extend deeply should be approached by a Krönlein incision.

ARNOLD SORSBY.

(7) **Kreibig, W. (Vienna).—Unusual ocular findings in multiple carcinomatous metastases. (Ueber ungewöhnliche Augenbefunde bei multiplen Karzinommetastasen).** *Zeitschr. f. Augenheilk.*, Vol. LXXIV, p. 362, 1931.

(7) **Kreibig** reports the case of a girl, aged 21 years, who died from multiple metastases due apparently to a bronchial carcinoma. There had developed bilateral oedema and exophthalmos, and post-mortem examination showed carcinomatous emboli in the chorio-capillaris and also extensive infiltration of both orbits with metastatic deposits. The emboli in the chorio-capillaris the author regards as blood-borne; their failure to develop is a well established characteristic of such cells when planted in a surrounding which for some reason in unfavourable to their further growth. In the extensive metastases in the orbits the author sees evidence in favour of the view that the orbit does indeed have a lymphatic supply.

ARNOLD SORSBY.

II.—MISCELLANEOUS

(1) **Harris, J. W. (Port Elizabeth).—Circumscribed abscess of the sclera.** *Jl. of the Med. Assoc. of South Africa*, p. 382, June 27, 1931.

(1) **Harris,** in his paper on circumscribed abscess of the sclera, comments on the fact that only in recent years has this condition been recognized as a separate clinical entity, and that it is not described in the standard text-books of ophthalmology.

The patient was a woman, aged 29 years, thin, anaemic and seven months pregnant. Six weeks before examination some hot fat had entered the left eye, but after a few days the eye recovered from the injury.

The abscess was situated between four and five o'clock, extended to the limbus and measured 5 mm. × 3 mm. × 2 mm., was soft and not tender on palpation. The conjunctiva was movable over the swelling. The swelling was incised and a large drop of thick green pus evacuated. Pieces of the neighbouring sclera were removed for examination. The underlying uvea was apparently unaffected. No micro-organisms were found in the pus, and the pieces of sclera were found to be composed of granulation tissue.

The author believes that the abscess was metastatic, but he was unable to detect any primary focus on clinical examination. He quotes cases from the literature.

H. B. STALLARD.

(2) **Hughes, Wendell L. (New York).—Spastic entropion: a simple procedure for its cure.** *Amer. Jl. of Ophthal.*, January, 1931.

(2) **Hughes** advocates the use of novocaine and alcohol for this condition. The technique is as follows: 0·3 c.c. of 4 per cent. novocaine is injected through the skin of the outer canthus into the fibres of the orbicularis muscle for a distance of about 4 to 5 mm. near the margin of the lower lid. Leaving the needle in place, the syringe is replaced by one containing 0·2 to 0·3 c.c. of 95 per cent. alcohol, which is injected into the same site. The results are apparent a few hours after the injection, which can be repeated in a few days, though this is seldom necessary. Five cases are reported in four of which the result was successful; the failure was in a patient where the tissues of the lower lid were very loose.

F. A. W-N.

(3) **Sabatsky, Kurt (Oels).—A new method for the rapid healing of ulcus serpens with retention of transparency of the cornea. (Eine neue Methode zur raschen Heilung des Ulcus serpens mit Erhaltung der Durchsichtigkeit der Hornhaut).** *Klin. Monatsbl. f. Augenheilk.*, Vol. LXXXIII, July-December, 1929.

(3) **Sabatsky** claims to have found in artificial wintergreen oil almost a specific remedy for abrasions, ulcers and opacities of the cornea. The treatment consists in scraping off, under cocaine anaesthesia, the foreign body or unhealthy tissue as the case may be, putting a tiny drop of wintergreen oil at the tip of a thin glass rod on the scraped area, gently rubbing it in, and then applying cocaine ointment—strength not mentioned—and bandaging the eye with "Elastoplast" or other bandage. Except for the scraping, which is not as a rule called for again, the treatment is repeated daily until healing is complete. The author has for years obtained practically transparent, smooth corneae in cases of corneal foreign bodies, abrasions and ordinary ulcers and this induced him to try the method in ulcus serpens. The treatment is the same as described above except that the scraped area is treated with one per cent. optochin solution before the wintergreen oil is rubbed in. Up to now he has treated 24 cases of pneumococcal ulcers, many of them very severe, and finds the method eminently satisfactory, the rapidity of cure with minimum of corneal change and retention of good vision constituting the outstanding features. He ascribes

the beneficial effect of artificial wintergreen oil to (1) its germicidal action and capacity to penetrate deeply into the corneal tissue; (2) its being almost a specific corneal tissue regenerator; (3) its tension-reducing action on the globe, sometimes rendering it so soft as to make one get the impression that perforation had occurred and (4) its capacity not only to clear existing scars and leucomata but also to influence new-forming corneal lamellae in such a way that from the outset no opaque tissue is formed.

The author adduces no laboratory evidence of the germicidal action of wintergreen oil. The trial of wintergreen oil in primary and secondary glaucoma to study its effect on intra-ocular pressure suggests itself to the reviewer.

D. V. GIRI.

(4) **Moore, Foster (London).—Cataract from exposure to X-rays.** *Lancet.* (Report of meeting of Section of Ophthalmology, Royal Society of Medicine, January 9, 1931). January 17, 1931.

(4) It seems advisable to notice in this journal the *Lancet* report of **Foster Moore's** case without waiting for the report of the R.S.M. During treatment of sycosis on the face heavy doses of X-rays were employed. The disease affected the eyelashes. The treatment lasted several months and the sycosis was completely cured. Sight began to fail between 1927 (the year of the treatment) and 1929. In June, 1930, the visual acuity was 6/60 in each eye. The visual defect was due to "a plaque-like opacity in the posterior cortex of each lens, while bordering this was a powdery opacity. The fundi were normal." The cataracts were successfully needled.

ERNEST THOMSON.

(5) **Nordmann, J. (Strasbourg).—Observations upon cataract in myotonia and endocrine cataract. (Considérations sur la cataracte myotonique et la cataracte endocrienne).** *Ann. d'Ocul.*, Vol. CLXVIII, p. 438, 1931.

(5) **Nordmann** describes in detail a case of typical atrophic myotonia affecting particularly the lower part of the face, the thenar and hypothenar muscles, the flexors of the fingers and the long supinators. The eyes and fields of vision were in all respects normal under ordinary examination. Vision with correction was 6/5 and 6/4. The corneal microscope, however, revealed bilateral cataract. In the region of the anterior and posterior zones of disjunction was a layer of numerous punctiform grey opacities with intermingled red and green crystals ("cristaux rouges et verts"). Between this layer and the capsule of the lens was an absolutely clear layer. The points of importance emphasized by Nordmann are: (1) The presence of the particular type of lens

opacities visible only by the corneal microscope, and causing no interference with vision; (2) The cataracts were found in a patient of 31 years of age, and were typical of cataracts due to endocrine disturbance. In addition to the well recognized features of precocity, bilaterality and superficiality of the cataracts, it was observed that there was a notable lack of demarcation between the several layers of the lens. This feature is present, therefore, in all types of endocrine cataract, diabetic, tetanic and myotonic. It is suggested that this sign may possibly be found before the development of opacities in the lens. (3) The antero-posterior diameter of the lenses was less than normal. This is a further sign present in the three groups of endocrine disturbance.

HUMPHREY NEAME.

(6) **Scotti, P. (Savona).—Changes in the lens caused by ultra-violet rays. (Alterazioni del Cristallino da Raggi Ultra-Violetti).** *Ann. di Ottal.*, December and January, 1931.

(6) Scotti has made a number of experiments in which the lens was exposed to ultra-violet rays; in some, enucleated eyes were used; in some, the lens extracted from the eye, and in others the living animal. The changes noted differed in the various classes of experiment; the enucleated eyes showed opacity of the nuclear zone of the lens. The living animals showed changes in the ciliary and capsular epithelium, and opacities in the lens chiefly close under the capsule.

That the opacity found in enucleated eyes is really due to the ultra-violet rays is shown by the fact that the interposition of a sheet of glass to stop these rays, prevented the formation of any opacity.

HAROLD GRIMSDALE.

BOOK NOTICES

Kurzes Handbuch der Ophthalmologie. Edited by F. SCHIECK and A. BRUCKNER. Band IV, pp. 874; 463 Figs. Berlin: Julius Springer. 1931. Price, R.M. 165.

The fourth volume of this large and extensive treatise on ophthalmology has now appeared, and follows the general lines adopted in its predecessors. The first part dealing with diseases of the conjunctiva, cornea, and sclera, has been contributed by Professor Schieck, of Würzburg. After dealing with the various types of infective conjunctivitis, in which are included concise and informative accounts of the bacteriology peculiar to each condition, the author adopts the somewhat novel and very useful arrangement

of grouping together a number of conditions which affect the conjunctiva and the corneal epithelium: inflammatory conditions, such as phlyctenular disease, acne rosacea, and so on; pigmentary changes; degenerations, in which are included pinguecula, keratosis, xerosis, and other conditions; and tumours. This is followed by a section dealing with pathological conditions peculiar to the cornea (developmental, inflammatory, degenerative) and a similar section on the sclera. The second part of the volume, written by Dr. Cramer, of Cottbus, is devoted to the effects of injuries to the eye; and this is followed by a section on sympathetic ophthalmitis, by Professor Reis, of Bonn. Here, as elsewhere in the book, the author contents himself with an encyclopaedic summary of different theories and views, and makes little attempt at a critical analysis which might lead to a reasoned course of treatment. The last section of the book—and the best—deals with the physiological and pathological variations of the intra-ocular pressure and the clinical aspects of glaucoma and buphthalmos. It is written by Professor Thiel, of Berlin, a recognized authority in this field, who bases his argument on the newer views that the aqueous humour is a dialysate. Our knowledge of the pathology of the various types of glaucoma is adequately summarized as well as the clinical symptoms of the disease, and the indications for medical and surgical treatment. Also the relative merits of the numerous surgical methods which have been advocated, are fairly reviewed.

Like its predecessors in this series the present volume is a valuable one, cumbersome perhaps in certain places, but on the whole well balanced. The illustrations are excellent and extremely informative, and like most German works of this type, the artistry of the production is beyond question. No effort is made to include an exhaustive literature, but that which is supplied is representative and useful.

An Index of Treatment. Edited by ROBERT HUTCHISON. 10th Edition, pp. 18 + 1027. Bristol: John Wright and Sons, Limited. London: Simpkin, Marshall, Limited. 1931. Price, 42s.

The ninth edition of this popular index was noticed in our columns in 1925 (Vol. IX, p. 543). It was edited by Hutchison and Sherren, and it was so quickly sold out that a reprint was brought out in December, 1926.

The present edition has been thoroughly revised; some of the articles have been rewritten and some new subjects are included, *inter alia*, Anaesthesia in Children, Diabetes in Childhood, Serum Sickness and Protein-shock Treatment.

The ophthalmic articles are again in the hands of Sir William Lister, a sufficient guarantee of their being accurate and up to date.

Recent Advances in Microscopy. By A. PINEY, BASIL GRAVES, E. W. MACBRIDE, H. R. HEWER, and E. C. BARTON-WRIGHT. London: A. Piney. 83 illustrations, pp. 254, 1931. Price, 12/6.

Microscopy is a wide subject and it would have been impossible for one man to write comprehensively on the advances which it has recently made. The volume under review is therefore the work of five authors, each of whom has taken a special aspect of his subject. The medical aspect is described by Piney, who is careful at the outset to emphasize the limitations of microscopy, *i.e.*, that histological methods can never do more than give us a picture equivalent to some living structures, and not a picture of these structures actually at work. Supra-vital staining suffers from the handicap that the structures made visible by this method are few in number, and that even those which are made visible are not shown in great detail. Piney's section opens with an interesting description of mitochondria, and then goes on to deal with the skin, blood, muscle and various glands. An interesting example of the complexity of modern histology is afforded by a paragraph on the subject of "that special form of histiocyte, the Kupffer cell, in the liver." It appears that there are at least three types of cells in the hepatic capillaries—the endothelial cells proper, branched pericytes between the endothelium and the liver cells and lastly, endocytes lying on the inner surface of the endothelium or even in the lumina of the capillaries. These last are presumably the true Kupffer cells.

The next section, on the microscopy of the living eye, directly concerns ophthalmologists and has been written by Basil Graves. He is to be congratulated on having given a remarkably clear and succinct account of the technique of this branch of ophthalmology within the compass of 57 pages.

The article contains 51 illustrations, which add greatly to the clarity of the descriptions in the text. Graves concerns himself mainly with technique, because the methods evolved for the examination of the human eye "are quite probably applicable in other useful fields, *e.g.*, zoological work; and also because in the space available a comprehensive account of common normal and pathological clinical features could not well be given." The various methods of examination—direct, proximal and retro-illumination, scleral scatter, etc., are described in considerable detail and a particularly good explanation is given of specular reflection.

The zoological aspect of modern microscopy is dealt with by MacBride and Hewer, who devote a large part of their space to a discussion and description of the Golgi apparatus. Work in this direction has been much facilitated lately by the perfection of an instrument for micro-dissection and by the discovery of tissue cultivation *in vitro*. The book concludes with a chapter on recent

advances in botanical microscopy by Barton Wright. Here again, the Golgi apparatus is to the fore, and there is an interesting account of the structure and behaviour of chromosomes. Micro-dissection also comes in for consideration, and it is interesting to read how the nucleus can be pushed by a delicate glass needle from end to end of a cell and then be watched, recoiling to its original position when released.

Although such a book as this is outside the confines of our speciality, it provides good reading and not the least of its uses, at any rate to ophthalmologists, is that it serves to remind them of the great progress which is being made in science as a whole as well as in that portion of it which concerns the structures within the walls of the orbit.

Augenärztliche Eingriffe. Ein Kurzes Handbuch für angehende Augenärtzte. By PROFESSOR DR. J. MELLER, Director of No. 1 Eye Clinic, Vienna. 3rd Edition. Vienna: Julius Springer. 1931. Price, 28 R.M., with duty and postage, 29·80.

The third edition of this book contains sixteen chapters, 352 pages including the index, and 199 illustrations, drawings and diagrams, demonstrating anatomical features, stages, and technical points in the surgery of the eye and its appendages. It has been revised and brought up to date and represents the technique employed in No. 1 Eye Clinic in Vienna, of which the author is professor and director.

Indications for operation, pre-operative preparation, surgical technique, post-operative treatment and complications are fully described. Difficulties encountered during operation are discussed and attention is paid to the faults that a beginner is likely to make and how such may be avoided. The stance of the operator, position of the assistant, and other technicalities are of the Viennese and continental schools and differ from those practised by the majority of English and American ophthalmic surgeons. These are clearly illustrated in the drawings (taken from photographs) and diagrams of operations. Methods of local anaesthesia, including retro-ocular injections and blocking of branches of the facial nerve are described. A chapter is devoted to reconstructive plastic surgery of the conjunctiva and lids, but this is brief and limited in the range of alternative operations for defects in these structures. Little is written about "whole skin" grafts and Thiersch grafts, or the methods of reconstructing the conjunctival fornix and contracted sockets.

The treatment of retinal detachment by ignipuncture has been added. The methods of localisation of the retinal tear are those practised in No. 1 Eye Clinic, Vienna. They are interesting and

elaborate, necessitating the use of a special type of perimeter and movable ophthalmoscope. When the meridian of the site of retinal hole is found further mathematical calculations as to its distance from the limbus are made by referring to the curves of a graph plotted for myopic, emmetropic, and hypermetropic eyes of various degrees. As a preliminary to the operation a small metal frame, which exactly encircles the limbus is made, and attached to this is a flat narrow strip of metal curved to the contour of the sclera. Two holes are bored in this ring at 3 and 9 o'clock respectively so as to lie in the transverse axis of the globe. The metal strip is set in the meridian of the retinal hole and is cut at such a length as to correspond to the distance it is believed to be from the limbus. Sutures are passed through the holes at 3 and 9 o'clock and these are used to secure the metal framework to the limbus. Cautery puncture is made at the point indicated.

This book is of considerable practical value to the ophthalmic practitioner. It is well written by a surgeon of great experience and the illustrations are clear and helpful,

A Pocket Atlas and Text-book of the Fundus Oculi. By G. LINDSAY JOHNSON, M.A., B.C., M.D.(Camb.), F.R.C.S.Eng. Second edition revised and enlarged. London: Adlard and Son, Limited. 1931. Price, 12/6.

This book has been carefully revised and brought up to date. Two chapters have been added, one on the various forms of cataract and another on the vitreous. There are numerous illustrations in the text, and fifty-four coloured drawings, selected from the author's cases and painted by Arthur W. Head, F.Z.S. Chapters are devoted to the ophthalmoscope, its history, varieties, and methods of use; to retinoscopy and refractive errors; and to the anatomy of the eye. Diseases that can be viewed by the ophthalmoscope are described.

There are 208 pages of text, and the coloured plates at the end of the book are accompanied by a description of the clinical aspects of the disease shown. These plates are valuable to the student for reference and to the teacher for demonstration purposes.

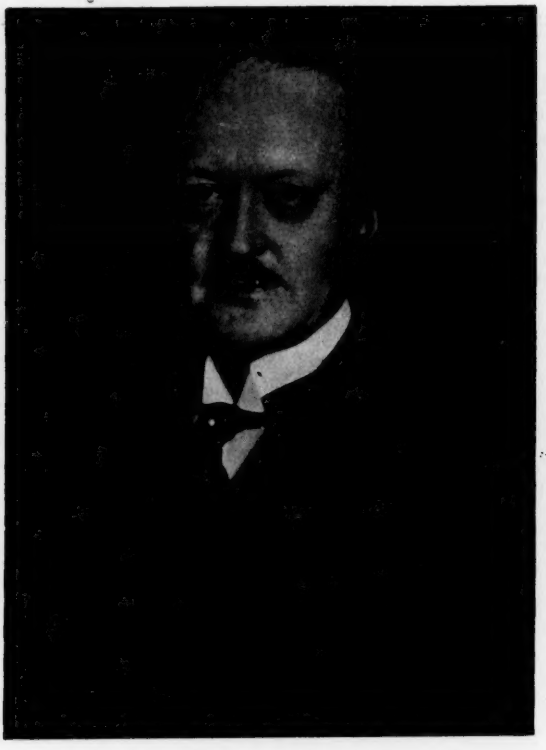
OBITUARY

PROFESSOR KONRAD KRISTIAN KARL LUNDSGAARD

We are indebted to DR. HARALD G. A. GJESSING, of Drammen, Norway, *for the following notice.*

NOT only the northern countries, but the whole ophthalmological world, has suffered a great loss by the death on August 30 of Professor K. K. K. Lundsgaard, in his 64th year.

The immediate cause of death was a secondary pneumonia, which terminated a long period of illness, which he bore with great fortitude.



Lundsgaard took his medical qualification in 1893 and thereafter spent some time in the Danish naval medical service. He then devoted himself to the study of bacteriology and surgery before taking up ophthalmology.

In 1900 he took his M.D. degree with a thesis on the relationship of the bacteriology of the conjunctiva to conjunctivitis. He then settled down in Copenhagen as an ophthalmic surgeon and started a private eye clinic, which soon acquired a great reputation. He also became attached to several hospitals, among others the Finsen Light Institute. Here he made a series of investigations on the light treatment of tuberculosis of the conjunctiva, of lupus of the eye and trachoma. These investigations were of great importance and spread his fame all over the world. So much so that some years ago his name was put on the list of those who were proposed for the professorship at the University Eye Clinic of Munich.

In 1925 Lundsgaard became Professor of Ophthalmology and Chief of the University Eye Clinic in Copenhagen. As those who heard his Doyne memorial lecture at Oxford in 1927 will realize, he was a splendid teacher and was able to arouse in his students great enthusiasm for his subject.

At the meeting of the International Ophthalmological Congress in Amsterdam in 1929 he was chosen to be President of the International Council of Ophthalmology, in succession to van der Hoeve.

Space does not permit me to give a full list of all Lundsgaard's publications, which number more than seventy, but the two chapters written by him in the "Nordisk Laerbok i Oftalmologi," those on diseases of the orbit and diseases of the conjunctiva can be quoted as examples of his large experience and remarkable power of clear exposition.

It is probable that the Scandinavian ophthalmologist will treasure most his last book, published in 1930, on "The different methods of making the operative section through the eyeball."

Lundsgaard was constantly interested in all new views and methods. He also invented and perfected many instruments, as for instance, his modified trephine and his portable dark room. This latter is of very great use in general hospital work. His chief interest, however, lay in the history of ophthalmology.

He was a very sociable man, an art connoisseur, especially of painting and a good and sincere friend. Those who had the privilege of his friendship have indeed suffered a great loss.

The *Acta Ophthalmologica* was founded in 1921 on his initiative, and he remained the chief editor. By this means he came personally into contact with all the Scandinavian ophthalmologists and he thus contributed in a large measure to a more intimate cultural union of the Northern nations

NOTES

Deaths

WE regret to record the death on October 10, of Robert Wallace Henry, the well-known ophthalmic surgeon of Leicester. Of Irish extraction, he was born in Dublin 64 years ago. He received his education at Trinity College, Dublin, and graduated B.A. in 1891; M.B., Ch.B., 1892, and M.D. in 1895.

After a period spent in Birmingham as Resident Superintendent of the Birmingham and Midland Eye Hospital, he began practice as an ophthalmic surgeon in Leicester in 1896, and quickly became recognized as an able exponent of his specialty. At the same time he began work for the British Medical Association, and held the post of Hon. Secretary of the Ophthalmic Section at the Leicester meeting in 1905. In 1912 he served as President of the Leicester Medical Society.

His reputation, already well known in the Midlands, was enhanced by the passing of the National Insurance Act, and it is a curious fact that, as in the case of the late Mr. E. B. Turner, Wallace Henry's reputation as a man of affairs and a mentor to thousands of his professional brethren, should have been suddenly, so to speak, brought to a climax by this piece of legislation.

The deaths are also announced of Professor Otto Haab, of Zürich, in his 81st year, and of Professor Golovine, of Moscow.

* * * *

Corrigenda

In the paper on papilloedema by Mr. Wolff and Mr. Davies in the November No., in the legend for Fig. 3, for "potassium ferrocyanide" read "Prussian blue," the actual injection fluid being the potassium ferrocyanide, ferric chloride mixture as explained in the text.

Dr. JOSEPH I. PASCAL writes to point out that we have misinterpreted his meaning on page 590, nine lines from the bottom of the page.

The word "monochromatic" should be substituted for "chromatic" aberration, and the sentence should read as follows:—

"A small allowance of about 0·5 D. should be made in the above calculations because of a so-called lag of accommodation due, according to Prof. Southall, to the monochromatic aberration of the eye."

We trust that Dr. Pascal will accept our apologies.

William Mackenzie Medal THE William Mackenzie Medal for 1931 has been awarded to Mr. Treacher Collins. The medal was presented at Glasgow on October 30, when Mr. Collins delivered an address, which will be published in this journal.

FUTURE ARRANGEMENTS

1931

December 1.—Midland Ophthalmological Society, at Birmingham.
December 4.—North of England Ophthalmological Society, at Leeds.
December 11.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting at the Royal Eye Hospital).

1932

January 8.—Section of Ophthalmology, Royal Society of Medicine.
February 5.—North of England Ophthalmological Society, at Newcastle-on-Tyne.
February 12.—Section of Ophthalmology, Royal Society of Medicine.
March 4.—North of England Ophthalmological Society, at Liverpool.
March 11.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting).
April 8.—North of England Ophthalmological Society, at Sheffield.
June 10.—Section of Ophthalmology, Royal Society of Medicine. (Annual Meeting).

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *October, 1931.*

ELLIOT. Some after-thoughts on trephining for glaucoma.
DAY, LANGSTON and O'BRIEN. Cataract and other ocular changes in vitamin *G* deficiency.
BLAKE. Spontaneous extrusion of intra-ocular foreign bodies.
EMMONS. The use of fascia lata in an extensive keratocele.
MAYER. Epinephrin in progressive myopia.
FERREE and RAND. The effect of relation to background on the size and shape of the form field for stimuli of different sizes.
EVANS and KERN. The relation of the parathyroid gland to cataract.
MUNNS. Accidental vaccination of the eyelids.
EDGERTON. A month's service with Dr. Holland at Shikarpur, India.

Archives of Ophthalmology. *September, 1931.*

JAMESON. The surgical entity of muscle recession.
BAILEY. Lesions of the cornea and conjunctiva in erythema exudativum multiforme (Hebra). Report of three cases with grave ocular sequelae.
PETER. Advancements and other shortening operations in concomitant squint.
MAYER. Ocular convergence. Its relation to basal metabolism and endocrine disease. A preliminary report.
KEY. Tuberculous keratitis. Descemetocele, perforation, repair. Report of a case.
SITCHEVSKA. An unusual corneal opacity. Report of a case.
BERLINER. Medullated nerve fibres associated with choroiditis. Report of a case with preliminary studies on the cause of the appearance of medullated nerve fibres in the retina.
GOLDSTEIN and WEXLER. Metastasis in the sheath of the optic nerve from carcinoma of the stomach.
FRALICK. Post traumatic tuberculous uveitis.

October, 1931.

GJESSING. Holth's iridencleisis antiglaucomatosa. Follow-up examination of 122 eyes from 6-159 months after operation.
YUDKIN. The presence of vitamin A in the retina.
FERREE, RAND and MONROE. Diagnostic scales for the 1 degree and 0·17 degree form field stimuli for the eight principal meridional quadrants taken separately.
HORINE and WEISS. The retinal changes of arteriosclerotic heart disease and essential hypertension.
SHANNON. Bitemporal paracentral scotoma. Report of a case.
BURKY and WOODS. Lens extract. Its preparation and clinical use.
HARTGRAVES and KRONFELD. Gunshot wound of the optic nerve. Report of a case.
FRIEDMAN. The development of the lens. Its significance in the interpretation of lenticular abnormalities.
BYERS. Buphthalmos secondary to congenital iridocyclitis.
KESTEN. Conjunctivitis and blepharitis due to yellow mercuric oxide.

Annales d'Oculistique. *September, 1931.*

GONIN. The results of obliterating cautery puncture of retinal tears.
CHARAMIS. Binasal hemianopia from pituitary tumour.

Archives d'Ophtalmologie. *October, 1931.*

VAN DUYSE. Heredity in ocular diseases.
MARTEL, MONBRUN and GUILLAUME. Acute papilloedema.
PESME. A case of myopic detachment of the retina successfully treated by Gonin's operation.
BOLOTTE and FRIBOURG-BLANC. A case of sanguinary tears from disturbance of the neuro-vegetative equilibrium following injury.
LAMPERT and LAMBERT. A rare case of migration of a foreign body in the vitreous.

Revue d'Oto-Neuro-Ophtalmologie. *October, 1931.*

BEAUVIEUX, DELMAS-MARSALET and DESPONS. Bilateral blepharospasm and alternating blepharospasm of encephalitic origin.
FAÇON, BRUCH and CHISER. Parinaud's syndrome with exaggeration of tonic reflexes in a case of haemorrhagic polio-encephalitis.
HORNING-WENGER. Peritonsillar abscesses and mydriasis.

Klinische Monatsblätter für Augenheilkunde. *September, 1931.*

VANNAS. A case of white dystrophic fat deposit in the periphery of the cornea.
WIBAUT. Studies on retinitis pigmentosa.
POOS. Clinical observations on a case of habitual spontaneous luxation of the globe.
SZÉKELY. On the appearance of lens substance in an abnormal position without visible disturbance of the aqueous.
RÖSSLER. On Apin's work on the use of the cobalt lamp for the estimation of refraction.
INOUYE. On the notification of axis in the combined clock and degree system.
ARCHANGELSKY and BRAUNSTEIN. On the pathological anatomy of ophthalmomyiasis interna.
FARKAS. On a rare triangular trefoil-shaped cataract with dehiscence of the anterior embryonic suture.
CHURGINA. Further experimental studies on the reversed effect of isolated tuberculous foci in skin and cornea.
HAUER. Blepharospasm as an early symptom of tabes dorsalis?
POPOVIC. Rheumatism and the eye.
DUDINOV. On the question of Salvati's treatment of squint.
WARSCHAWSKI. On the question of the removal of the tarsal plate in trachomatous entropion.
LÖWENSTEIN. On the genesis of congenital cataract.
——— On the indications and technique of Denig's mucous membrane graft.
WEINSTEIN, On the question of the physico-chemical mechanism of cataract.
KRAUPA. Heat destruction of the lens capsule.
VOGT. Answer to Kraupa's previous procedure.

October, 1931.

KUNZ. Abnormal size of the lens as a cause of adolescent glaucoma.
BRAUN. A new hemikinesimeter.
SONDERMANN. On the interpretation of the intra-ocular tension in healthy and diseased eyes.
BIETTI. On a melanocarcinoma arising from pigment epithelium in the globe and orbit.
VOM HOFE and HARTUNG. Further experimental observations on hypertonus following hypotonus.
HOMMEL. Nodular iritis in diabetes insipidus.
ROLETT. Statistics of tuberculous inflammation of the eye from the University clinic of Berne.
DONATH. Observations on the paper by Dr. A. Petrovic and Prof. Dr. Alex. Tschemolossow "On the question of voluntary dilatation of the pupil."
KRAUPA. "The vitreous point" (Litinsky).

Archivio di Ottalmologia. *July-August, 1931.*

ROSSI. Heterochromia of the iris and tuberculous affections.
GALLENGA. Synchisis scintillans in the anterior chamber with special regard to its pathogenesis.
SCALINCI. The ocular operations of Benvenuto *(continued)*.
FERRARI. Sub-capsular stationary opacity of the lens from direct contusion.
LUGLI. On marginal degeneration of the cornea.

Bollettino d'Oculistica. *August, 1931.*

LO CASCIO and RINALDI. Researches on so-called phako-anaphylactic endophthalmitis.
SALVIOLI. My apparatus for the study of the superficial capillaries of the eye.
GUALDI. Experimental researches and clinical observations on the behaviour and the mechanism of the pupil reactions in convergence.

SALA. Contribution to the study of marginal degeneration of the cornea.
BUSSOLA. Ivory exostosis and medullary exostosis of the orbital walls.
NICHELATTI. Clinical and histological contribution to the study of certain rare tumours of the lids.

September, 1931.

CHARLIN. The syndrome of the nasal nerve.
MARGOTTA. Researches on dazzling.
ACCARDI Alterations of the iris in chicken pox.
LAMMA. On the connection between angiomata of the face and alterations in the fundus oculi.
BUSSOLA. Bilateral metastatic pneumococcal ophthalmia from lung infection.
MOTOLESE. Pseudo-albuminuric retinitis of fleeting development.

Lettura Oftalmologica. *August, 1931.*

MONTANELLI. Cataract from lightning.
SANNA. Persistence of a piece of steel in the eye with partial conservation of vision and absence of siderosis.
BAQUIS. Polypi of the lacrimal canaliculi.

Revista Oto-Neuro-Oftalmologica. *July-August, 1931.*

NEUSCHÜLER. On thrombo-phlebitis of the cavernous sinus with the rare event of an intraventricular cerebral haemorrhage.
PETRAGNANI. Left homonymous hemianopia from calcification of the internal carotid.
MOSCARDI. Paresis of the superior oblique in indirect cranial trauma.

Archivos de Oftalmologia Hispano-Americanos.

October, 1931.

LUQUE. Solitary retinal hypertension from meningo-encephalitis.
CARNICER. Contribution to the anatomico-pathological study of orbital melanomata.
CAMISÓN. Value of bacteriological examination of the conjunctiva as a preliminary to operation.
MENDOZA. Treatment of relapsing pterygium.

Revista Oto-Neuro-Oftalmologica y de Cirugia Neurologica.

October, 1931.

MAGITOT. The aqueous in glaucoma.
TEULIERES and BEAUVIEUX. Late ocular manifestations in epidemic encephalitis.
VANRELL. Synthesis.
MATTOS. Colour photography of the fundus of the human eye.
PAVIA. Retinography in colours. First experiments in direct photography of the fundus with autochrome films.

November, 1931.

PAVIA. Retinography in colours First experiments in direct photography of the fundus with autochrome films *(continued)*.
BAILLIART. The oscillations of the needle of the tonometer.
MONIZ. Arterial encephalography.

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